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THE BOSTON
Rec. Aug. 26, 1907.
Medical and Surgical
JOURNAL.

TRANSACTIONS

OF THE

AMERICAN PEDIATRIC SOCIETY

EIGHTEENTH SESSION

HELD AT HOTEL TRAYMORE, ATLANTIC CITY, N. J.,
MAY 30, 31 AND JUNE 1, 1906

EDITED BY
LINNÆUS EDFORD LA FÉTRA, M.D.

VOLUME XVIII.

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PRESIDENTS.

1889. A. Jacobi, M.D.
1890. J. Lewis Smith, M.D.
1891. T. M. Rotch, M.D.
1892. Wm. Osler, M.D.
1893. A. D. Blackader, M.D.
1894. John M. Keating, M.D.
1895. F. Forchheimer, M.D.
1896. Joseph O'Dwyer, M.D.
1897. Samuel S. Adams, M.D.
1898. L. Emmett Holt, M.D.
1899. Wm. P. Northrup, M.D.
1900. Henry Koplik, M.D.
1901. Wm. D. Booker, M.D.
1902. W. S. Christopher, M.D.
1903. J. P. Crozer Griffith, M.D.
1904. Augustus Caillé, M.D.
1905. C. G. Jennings, M.D.
1906. A. Jacobi, M.D.
1907. B. K. Rachford, M.D.

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F. S. Churchill, M.D.
L. Emmett Holt, M.D.
John Lovett Morse, M.D.
S. McC. Hamill, M.D.

MEETING PLACES.

1888. Washington, D. C. (Organization), September 18.
1889. Washington and Baltimore, September 20 and 21.
1890. New York, June 3 and 4.
1891. Washington, September 22 and 25.
1892. Boston, May 2, 3 and 4.
1893. West Point, N. Y., May 24, 25 and 26.
1894. Washington, May 29 and June 1.
1895. Virginia Hot Springs, May 27, 28 and 29.
1896. Montreal, May 25, 26 and 27.
1897. Washington, May 4, 5 and 6.
1898. Cincinnati, June 1, 2 and 3.
1899. Deer Park, June 27, 28 and 29.
1900. Washington, May 1, 2 and 3.
1901. Niagara Falls, May 27, 28 and 29.
1902. Boston, May 26, 27 and 28.
1903. Washington, May 12, 13 and 14.
1904. Detroit, May 30, 31 and June 1.
1905. Lake George, N. Y., June 19, 20 and 21.
1906. Atlantic City, N. J., May 30, 31 and June 1.
1907. Washington, May 7, 8 and 9.

OFFICERS, 1907.

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First Vice-President.....WALTER LESTER CARR, M.D.
Second Vice-President.....DAVID L. EDSALL, M.D.
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JOHN LOVETT MORSE, M.D.
S. McC. HAMILL, M.D.
GEORGE N. ACKER, M.D.

MEMBERS.

ABT, ISAAC A., M.D.....4326, Vincennes Avenue, Chicago
ACKER, GEORGE N., M.D.....913, Sixteenth Street, Washington
ADAMS, SAMUEL S., M.D.....1, Dupont Circle, Washington
BAINES, ALLEN, M.D.....194, Simcoe Street, Toronto
BLACKADER, A. M., M.D.....236, Mountain Street, Montreal
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DORNING, JOHN, M.D.....111, West Forty-eighth Street, New York
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EATON, PERCIVAL J., M.D.....131, N. Highland Avenue, E. E., Pittsburgh
EDSALL, D. L., M.D.....1432, Pine Street, Philadelphia
FORCHHEIMER, F., M.D.....Fourth and Sycamore Streets, Cincinnati
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GRAHAM, E. E., M.D.....1713, Spruce Street, Philadelphia
GRIFFITH, J. P. CROZER, M.D.....1810, Spruce Street, Philadelphia

HAMILL, S. McC., M.D.....1822, Spruce Street, Philadelphia
HAND, ALFRED, JR., M.D.....1724, Pine Street, Philadelphia
HOLT, L. EMMETT, M.D.....14, West Fifty-fifth Street, New York
HOWLAND, JOHN, M.D.....49, East Fifty-third Street, New York
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KOPLIK, HENRY, M.D.....692, Madison Avenue, New York
LADD, MAYNARD, M.D.....220, Marlborough Street, Boston
LA FÉTRA, LINNÆUS E., M.D.....58, West Fifty-eighth Street, New York
MCCOLLOM, JOHN H., M.D.....745, Massachusetts Avenue, Boston
MARTIN, C. F., M.D.....33, Durocher Street, Montreal
MILLER, D. J. MILTON, M.D.....1801, Pine Street, Philadelphia
MORSE, J. LOVETT, M.D.....70, Bay State Road, Boston
NORTHRUP, WILLIAM P., M.D.....57, East Seventy-ninth Street, New York
PUTNAM, CHARLES P., M.D.....63, Marlborough Street, Boston
RACHFORD, B. K., M.D.....323, Broadway, Cincinnati
ROTCH, THOMAS MORGAN, M.D.....197, Commonwealth Avenue, Boston
RUHRÄH, JOHN, M.D.....839, North Eutaw Street, Baltimore
SAUNDERS, E. W., M.D.....3003, Lafayette Avenue, St. Louis, Mo.
SHAW, HENRY L. K., M.D.....198, Washington Avenue, Albany
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SOUTHWORTH, THOMAS S., M.D.....807, Madison Avenue, New York
STARR, LOUIS, M.D.....1818, South Rittenhouse Square, Philadelphia
TOWNSEND, CHARLES W., M.D.....76, Marlborough Street, Boston
WENTWORTH, A. H., M.D.....352, Marlborough Street, Boston
WEST, J. PARK, M.D.....3608, Belmont Street, Bellaire, Ohio
WESTCOTT, THOMPSON S., M.D.....1833, Spruce Street, Philadelphia
WILLIAMS, HAROLD, M.D.....528, Beacon Street, Boston
WILSON, W. REYNOLDS, M.D.....1709, Spruce Street, Philadelphia
WINTERS, J. E., M.D.....25, West Thirty-seventh Street, New York

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DR. JOHN THOMPSON.....Edinburgh, Scotland
DR. GEORGE F. STILL.....London, England
DR. HENRY ASHBY.....Manchester, England
DR. O. HEUBNER.....Berlin, Germany
DR. THEODOR ESCHERICH.....Vienna, Austria
DR. WILLIAM OSLER.....Oxford, England
DR. A. BAGINSKY.....Berlin, Germany
DR. V. HUTINEL.....Paris, France

Deceased.

JOHN A. JEFFRIES, M.D.
Born, September 2, 1859,
Died, March 26, 1892.

THOMAS F. SHERMAN, M.D.
Born, March 17, 1856,
Died, September 26, 1893.

JOHN M. KEATING, M.D.
Born, April 20, 1852,
Died, November 17, 1893.

CHARLES WARRINGTON EARLE, M.D.
Born, 1845,
Died, November 19, 1893.

J. LEWIS SMITH, M.D.
Born, October 15, 1827,
Died, June 9, 1897.

JOSEPH O'DWYER, M.D.
Born, October 12, 1841,
Died, January 7, 1898.

JOHN HENRY FRUITNIGHT, M.D.
Born, November 9, 1851,
Died, December 18, 1900.

FREDERICK A. PACKARD, M.D.
Born, November 17, 1862,
Died, November 1, 1902.

WALTER S. CHRISTOPHER.
Born, 1859,
Died, March 2, 1905.

LEROY MILTON YALE.
Born, February 12, 1841.
Died, September 12, 1906.

Obituary.

LEROY MILTON YALE, M.D.

Dr. Leroy Milton Yale was born at Holmes Hole (now Vineyard Haven), Mass., February 12, 1841, the son of Leroy Milton and Maria Allen Yale, and died September 12, 1906. He graduated from Columbia College in 1862, and from Bellevue Hospital Medical College in 1866. In that institution he lectured for some time on orthopedic surgery. For a few years he held a lectureship on obstetrics in the University of Vermont. He was surgeon to Charity Hospital from 1870 to 1877, to Bellevue Hospital from 1877 to 1882, to Presbyterian Hospital from 1880 to 1885. He was a member of the New York County Society and Academy of Medicine, and, since 1891, of the American Pediatric Society, in which, though contributing but little beyond his paper on the Local Treatment of Diphtheria contained in the fifth volume of its transactions, he was greatly interested. He was co-editor of the *Medical Gazette,* 1867-68, and medical editor of *Babyhood,* 1884-1891. His books are: "Nursery Problems," New York and Philadelphia Contemporary Publication Company, 1893, pp. 274, 12mo, and "The Century Book of Mothers," 1901. His journal articles quoted in the subject Catalogue of the Surgeon General's Library are as follows:

"A Report of the Epidemic of Cholera Occurring on Blackwell's Island, New York, in the Summer of 1866." Pp. 109-197. New York: F. McElroy, 1867. (Reprint from Annual Report, Commissioner Charities and Corrections, New York, 1867.)

"The Argument for the Antipyretic Treatment of Fever." Pp. 20, 8vo. New York: D. Appleton & Co., 1874. (Reprint from *New York Medical Journal,* 1874, Vol. XX.)

"Phimosis; A Report from the Surgical Section of the New York Academy of Medicine." Pp. 15, 8vo. D. Appleton & Co., 1877. (Reprint from *New York Medical Journal,* 1877, Vol. XXVI.)

"Certain General Considerations Respecting the Mechanical Treatment of Chronic Diseases of the Joints, with Special Reference to the Use of Traction." Pp. 16, 8vo. Trow & Co., 1878. (Reprint from *Medical Record,* 1878, Vol. XIII.)

"The Academy as a Teacher." An Anniversary Address De-

livered before the New York Academy of Medicine, December 11, 1879. Pp. 19, 8vo. New York, 1879.

"Remarks on Excision of the Hip." Pp. 15, 16mo. New York, 1885. (Reprint from *New York Medical Journal,* 1885, Vol. XLII.)

"Some Notes Concerning Measles." *Babyhood,* 1896, Vol. XII., pp. 129-131.

"Concerning Phimosis." *Babyhood,* 1896, Vol. XII., 314-316.

"Constipation in Childhood and its Domestic Management." *Babyhood,* 1897, Vol. XIII., pp. 31-34.

"On the Care and Modification of Milk for Infants' Use." *Canada Lancet,* 1897-98, Vol. XXX., 487-497.

"Overstudy." *New York Medical Journal,* 1900, Vol. LXXI., pp. 225-227.

"The Diagnosis of Early Hip Joint Disease from Rheumatism, Neuralgia and So-called 'Growing Pains.'" *American Medico-Surgical Bulletin,* May, 1893.

Being much interested in outdoor sports and especially in fishing, he wrote various magazine articles, several of which were published in *Outdoor Library.* ("Angling.")

But that was not all there was to our quiet, bright and versatile friend. He was credited by good judges with an unusual knowledge of the technical processes of the art of etching. In the establishment of the New York Etching Club he took an active part, and was its president from 1877-79. From the pen of Mr. Frank Weitenkampf, Curator of Prints at the New York Public Library, we have the following statement: "Dr. Yale produced several hundred plates, mostly landscapes, of which a certain number were in the nature of an artistic traveler's memoranda of places visited. The best of his serious work had the qualities demanded of painter-etching and found in the plates of the masters of the art—good composition, economy of line and a proper sense of both the possibilities and limitations of the etching-needle. Dr. Yale was, furthermore, an enthusiast on the subject of etching, who encouraged more than one to take up the needle. His name is indissolubly connected with the development of painter-etching in this country, and he took an active part in the founding of the New York Etching Club.

"He, James D. Smillie and the late R. Swain Gifford naturally come to mind together, and these three in fact were associated in

the production of the little plate etched at the first meeting of the New York Etching Club, in 1877, Yale working the press. The original copper plate of this etching is in the print room of the New York Public Library, which institution possesses also a number of etchings by Dr. Yale selected by himself and representing his best work. Dr. Yale took an active interest in the growth of the library's collection of American etchings, gave a manuscript catalogue of his work, as well as one or two original coppers and a number of etching tools, and urged others to follow his example. In recent years he found much pleasure in making pastel studies of landscape, but as an artist he will remain best known by his etching work. His is an influential figure in the history of painter-etching in this country." That is why only lately the July number of the *Bulletin of the New York Public Library* counts up his name with those of J. Alden Weir, R. Swain Gifford, E. D. French, F. S. King, Henry Wolf, T. R. Sugden, and Geo. N. Boughton.

On as late a date as February 8, 1906, Dr. Yale wrote in his "List of Etchings, by Leroy Milton Yale, 238 plates now in the Lenox Library," the following statement:

"They are not to be taken seriously, as a majority are merely slight sketches on copper, it having for many years been my habit to carry in my pocket a prepared copper in place of a sketch book, and to occupy moments of detention with etching anything at hand.—L. M. Yale."

This somewhat elaborate reference to Dr. Yale's non-medical accomplishments will surprise only those whose ambitions and attainments are confined within the limits of professional study and practice. It is only the average mind that is satisfied with a single study, and most men's vision is limited by a narrow horizon. Still, we have gradually become accustomed to look up with admiration and approval to the few extraordinary men who combine medicine and literature in a rare degree. Their greatness has become our pride. Medical men, however, who have excelled in art like Dr. Yale are very rare indeed. His example should teach us that a proficient physician may be a master in other fields as well, and that in the very best of our race many and different sciences and arts may be combined to create a man whose erudition and practical work will be blended into a perfect ethical and esthetical harmony.

His medical attainments were not always estimated at their full value, though he had enjoyed opportunities to gather knowledge and attain proficiency to a rare degree. His early surgical career taught him accuracy and care in all his work, though he may gradually have shrunk from mechanical labor. But thoughtful surgeons are certain to become good diagnosticians. Their habits of precision he carried into the general practice of his later life, and he became a good practitioner in the best sense of the word, the very philosophical physician whom Plato delights in calling godlike. His heart was the equal of his brain. The kindly and lovable traits of his nature were exhibited both in his daily life and his professional work. That is what attracted him to children, and children to him. That, and his general information, acquired through the habit of utilizing every moment of his life, a good part of which was blighted by infirmities quietly borne, made him a delightful companion. Moreover, no man could be more averse to the prevailing commercial spirit of our period than Dr. Yale. Altogether, he was one of those who, no matter at what age, always die too soon. A. J.

LEROY MILTON YALE, M.D.

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MINUTES OF THE EIGHTEENTH ANNUAL MEETING OF THE AMERICAN PEDIATRIC SOCIETY.

Held at Hotel Traymore, Atlantic City, N. J., on May 30, 31 and June 1, 1906.

The following members were present: Doctors Isaac A. Abt, Chicago; George N. Acker, Washington; Samuel S. Adams, Washington; Walter Lester Carr, New York; Henry Dwight Chapin, New York; F. S. Churchill, Chicago; A. C. Cotton, Chicago; John Dorning, New York; Percival J. Eaton, Pittsburgh; David L. Edsall, Philadelphia; Rowland G. Freeman, New York; E. E. Graham, Philadelphia; J. P. Crozer Griffith, Philadelphia; S. McC. Hamill, Philadelphia; Alfred Hand, Jr., Philadelphia; L. Emmett Holt, New York; Francis Huber, New York; A. Jacobi, New York; J. H. Mason Knox, Baltimore; Henry Koplik, New York; Linnæus E. La Fétra, New York; J. Lovett Morse, Boston; William P. Northrup, New York; B. K. Rachford, Cincinnati; Thomas Morgan Rotch, Boston; John Ruhräh, Baltimore; Irving M. Snow, Buffalo; Thomas S. Southworth, New York; Louis Starr, Philadelphia; A. H. Wentworth, Boston; J. Park West, Bellaire; Thompson S. Westcott, Philadelphia; W. Reynolds Wilson, Philadelphia; J. E. Winters, New York; Leroy M. Yale, New York.

The following visitors were present: Doctors Malcolm Goodridge, New York; Henry E. Hale, New York; J. H. Hess, Chicago; J. Roberts Johnson, Syracuse; Philip Marvel, Atlantic City; F. S. Meara, New York; Charles H. Miner, Wilkesbarre; Albert William Myers, Milwaukee; A. N. Richards, New York.

FIRST SESSION, MAY 30, 10 A.M.

The meeting was called to order by the president, Dr. A. Jacobi. The minutes of the Seventeenth Annual Meeting were adopted as published in the ARCHIVES OF PEDIATRICS.

1. Dr. Thomas Morgan Rotch, of Boston, with the collaboration of Dr. Maynard Ladd and Dr. Charles Hunter Dunn, pre-

sented a paper entitled "Suggestions as to the Classification of Gastroenteric Diseases on an Etiological Basis."

Discussion by Drs. Holt, Rachford, Jacobi, Griffith, Freeman and Rotch.

2. Dr. L. Emmett Holt, of New York, read a paper on "Some Phases of the Feeding Problem."

Discussion by Dr. Griffith, who exhibited a glass graduate for percentage modification of milk, and by Drs. Adams, Northrup, Graham, Southworth, Morse, La Fétra, Rotch, Cotton, Abt, Westcott, Churchill, Winters, Freeman, Jacobi and Holt.

3. Dr. J. Lovett Morse, of Boston, read a paper on "Acidified Milk in Infant Feeding."

Discussion by Drs. Graham, Southworth, Abt, Jacobi and Morse.

4. Dr. David L. Edsall, of Philadelphia, presented a paper entitled "Observations Concerning the Nature of Atrophy of Intestinal Origin."

Discussion by Drs. La Fétra, Wentworth, Winters and Edsall.

5. Dr. Thompson S. Westcott, of Philadelphia, presented a paper on "Modified Wet-Nursing."

Discussion by Dr. Cotton.

SECOND SESSION, MAY 30, 3 P.M.

6. Dr. Rowland G. Freeman, of New York, read a paper entitled "The Relationship of Weight and Length in Infancy."

7. Dr. J. P. Crozer Griffith and Dr. J. Claxton Gittings, of Philadelphia, presented a paper on "Weight in the First Two Weeks of Life."

Discussion of Dr. Freeman's and Dr. Griffith's papers by Drs. Churchill, Holt, Koplik, Rotch, Winters, Jacobi, Chapin, Griffith and Freeman.

8. Dr. Irving M. Snow, of Buffalo, presented "A Case of Primary Tuberculous Infection of the Intestine" (specimen).

Discussion by Drs. Rachford, Northrup, Rotch, Adams, Westcott, Holt, Hamill, Hand and Snow.

9. Dr. Linnæus E. La Fétra, of New York, reported a case of "Tuberculous Cervical Adenitis in a Four Months Infant."

Discussion by Drs. Koplik, Cotton and Hess (visitor).

10. Dr. Alfred Hand, Jr., of Philadelphia, reported "A Case

of Tuberculous Pericarditis, with Frequent Aspiration of Fluid from the Pericardial Sac. Death from Tuberculous Meningitis."

Discussion by Drs. Griffith, Abt, Koplik, Rotch, Morse, Edsall and Hand.

11. Dr. Henry D. Chapin, of New York, read a paper entitled "The Teaching of Scientific Infant Feeding. A Plea for More Uniformity."

Discussion by Drs. Cotton, Southworth and Chapin.

12. Dr. William P. Northrup, of New York, presented a paper on "The Twenty-three Hour Treatment."

Discussion by Drs. Cotton, Graham, Hand, La Fétra, Winters and Northrup.

THIRD SESSION, MAY 31, 9:30 A.M.

13. Dr. A. Jacobi delivered the presidential address, entitled "The Tonsil as a Portal of Microbic and Toxic Invasion."

14. Dr. Henry Koplik, of New York, read a paper with the title, "'Anemia Infantum Pseudoleukemica' of von Jaksch. A Contribution to the Pathology, Hematology and Clinical History of the Affection."

Discussion by Drs. Morse, Churchill, Hamill, Hand, Griffith and Koplik.

15. Dr. A. C. Cotton, of Chicago, read a paper on "Sepsis in the Newly-born, with Postmortem Reports."

Discussion by Drs. Hamill, Edsall, Rotch, Koplik, Holt, Jacobi and Cotton.

16. Dr. B. K. Rachford, of Cincinnati, reported "A Case of Septic Endocarditis."

17. Dr. Alfred Hand, Jr., of Philadelphia, reported "A Case of Septic Endocarditis following Suppurative Otitis Media."

Discussion of Dr. Rachford's and Dr. Hand's papers by Drs. Rotch, Koplik, Yale, Edsall, Morse, Churchill and Hand.

18. Dr. S. McC. Hamill, of Philadelphia, with the collaboration of Dr. Theodore Le Boutillier, presented a paper entitled "Inorganic Late Systolic Pulmonary Murmurs in Infancy and Childhood."

Discussion by Drs. Northrup, Chapin, Ruhräh, Abt, Morse, Edsall, Freeman, Rotch, La Fétra, Holt and Hamill.

19. Dr. J. H. Mason Knox, of Baltimore, in collaboration

with Edwin H. Schorer, presented a paper on "Rhabdomyomata of the Heart."

20. Dr. Rowland G. Freeman, of New York, exhibited "A Glass Graduate for Measuring Milk Sugar."

FOURTH SESSION, MAY 31, 3 P.M.

21. Dr. Francis Huber, of New York, demonstrated a pocket portable "Siphon Aspirator."

22. Dr. Thomas Morgan Rotch, of Boston, presented "Some Specimens of Foreign Bodies in the Bronchi and Intestines of Children."

Discussion by Drs. Northrup, Holt, Koplik, Graham, Eaton, Huber, Adams, Hamill, Griffith and Edsall.

23. Dr. L. Emmett Holt, of New York, presented photographs of "Two Cases of Bromid Eruption."

24. Dr. Percival J. Eaton, of Pittsburgh, read two papers: (*a*) "A Case of Measles Superimposed on Scarlatina"; (*b*) "Koplik's Sign in a Recent Epidemic of Measles."

Discussion by Drs. Koplik, Graham, Adams, Acker, Freeman and Eaton.

25. Dr. S. McC. Hamill, of Philadelphia, read a paper entitled "Area of Impaired Resonance Behind and Beneath the Inner Third of the Left Clavicle."

Discussion by Dr. Jacobi.

26. Dr. I. A. Abt, of Chicago, presented a paper on "Acute Nonsuppurative Encephalitis in Children."

Discussion by Drs. Morse and Griffith.

27. Dr. B. K. Rachford, of Cincinnati, reported "A Case of Hydrencephalocele and Spina Bifida."

28. Dr. Samuel S. Adams, of Washington, reported "A Case Simulating Peptic Ulcer."

EVENING SESSION.

29. Dr. Rowland G. Freeman, of New York, gave a talk on "The Influence of Milk Certification on Dairy Hygiene, Illustrated by Lantern Slides."

A vote of thanks was given to Dr. Freeman for his illustrated lecture.

The remainder of the evening was devoted to a most enjoy-

able "smoker" given the Society with the compliments of the president, Dr. Jacobi.

FIFTH SESSION, JUNE 1, 9 A.M.

30. Business meeting. The report of the Council was presented, and upon its recommendation the following actions were taken:—

The proposition of E. B. Treat & Co. in regard to the publication of the Transactions of the Society was accepted.

The following officers were elected for the ensuing year:—president, Dr. B. K. Rachford; first vice-president, Dr. Walter Lester Carr; second vice-president, Dr. David L. Edsall; secretary, Dr. Samuel S. Adams; treasurer, Dr. J. Park West; recorder and editor, Dr. Linnæus E. La Fétra.

Dr. George N. Acker was elected a member of the Council to take the place of Dr. J. P. Crozer Griffith, whose term expired.

Dr. A. Jacobi was elected a member of the Executive Committee of the Congress of American Physicians and Surgeons. Dr. W. P. Northrup was elected as alternate.

Dr. Samuel S. Adams was elected a member of the Committee of Arrangements of the Congress.

The Society voted to hold the next meeting in Washington, D. C., in May, 1907, at the time of the Congress of American Physicians and Surgeons.

Dr. Charles Hunter Dunn, of Boston, was elected to membership.

The report of the treasurer was presented, and Drs. Freeman and Carr were appointed a Committee of Audit.

The annual dues were fixed at ten dollars.

A rising vote of thanks was given to the management of the Hotel Traymore for their courtesies.

31. Dr. Thomas S. Southworth, of New York, reported a case of "Typhoid Infection Conveyed by a Convalescent Infant."

Discussion by Dr. Edsall.

32. Dr. Henry Koplik, of New York, in collaboration with Dr. Henry Heiman, presented "A Study of Relapses in the Typhoid Fever of Children."

Discussion by Drs. Rotch and Abt.

33. Dr. John Howland, of New York, in collaboration with

A. N. Richards, Ph.D., presented a paper entitled "A Consideration of Some Possible Etiological Factors in Recurrent Vomiting."

Discussion by Drs. Griffith, Edsall, Freeman and Richards (by invitation).

34. Dr. A. C. Cotton, of Chicago, read a paper on "Gonorrhea in Infants."

35. Dr. Isaac A. Abt, of Chicago, presented "A Note on the Reducing Power of Urine, Following the Administration of Urotropin."

36. Dr. George N. Acker, of Washington, reported 2 cases of "Carbolic Acid Poisoning by Rectal Injections."

Discussion by Dr. Adams.

The following papers were read by title:—

1. "A Study of the Stools of Breast-fed Infants in Relation to the Presence of Starch Enzyme," by Dr. Charles G. Kerley, of New York, in collaboration with Drs. H. A. Craig and H. N. Mason.

2. "Unusual Temperatures in Pneumonia," by Dr. Francis Huber, of New York.

A vote of thanks was given to the president for a very successful meeting.

LINNÆUS EDFORD LA FÉTRA,
Recorder.

THE TONSIL AS A PORTAL OF MICROBIC AND TOXIC INVASION.

BY A. JACOBI, M.D., LL.D.,
New York.

You have been good enough to re-elect me to the position I occupied during the early part of the existence of this Society. The honor, for which I thank you from the innermost of my heart, was surely unexpected; for a re-election under these circumstances is unusual. Let me now, however, that I have been the recipient a second time of this great distinction, suggest to you the possibility of abolishing the old custom of never according a re-election to a former incumbent. There may be reasons for not changing it, but I can imagine that the Society may not forever bind itself to persist in annual changes. The desire to become the presiding officer of this distinguished body is justifiable and natural, but it may be in its occasional interest to reappoint a former officer.

My present remarks should not be considered in the light of a formal address. Nothing I could present to you satisfies my wish to serve you; but the exigencies of the past weeks have prevented me from coming before you with an essay worthy of the occasion. Still, it occurred to me that you would be satisfied with the consideration of a problem which appears to become less soluble the more copious its literature has grown. What I allude to is the rôle the tonsil plays in the absorption of bacteria or toxins. Clinical observations on that subject are very numerous, and deserve the same credit due to every accurately observed fact; but will rarely be numerous enough to decide the exclusive or partial port of entry of a poison into the circulation. For we are seldom in a position to separate the tonsil from its neighborhood during the acute invasion or the rapid progress of a microbic or toxic poison. Altogether, I mean to approach the subject as well from an anatomical point of view as from that of clinical experience.

At the head of what I have to say I place observations I have made many hundreds of times, formulated in papers and books repeatedly these forty and more years, and which have been confirmed by the observations of others. Cases of membranous throat diseases behave differently according to their location. What we called diphtheria in 1858 and twenty-five years afterwards and at present either diphtheria, or pseudodiphtheria, or coccus invasion, or what not, behaves alike in its relation to the lymph system and especially to the lymph bodies of the neighborhood. The observations are easy, because in these affections there is a visible result of the invasions in the shape of membranous tufts or layers, and the invasion itself can be traced to an almost always definite day followed by a brief incubation, contrary to what we know of the invasion of tuberculosis. Now, whenever the membrane is limited to the tonsil there is very little or no glandular swelling in the neighborhood. On the other hand, if a membrane extends from the tonsil to its neighborhood, or starts at a distance from the tonsil, neighboring lymph bodies swell at once. Again, the treatment of this neighborhood shows its effect almost immediately on the swelled glands. That is mostly evident when the seat of the membrane is anywhere in the posterior nares which excel by an immense network of lymphatics. On the other hand, when it covers the vocal cords and Morgagni's fossa, both of which have only a scanty network of lymphatics, there is no adenitis nor any constitutional symptoms. Indeed, it has been well known these thirty years that an uncomplicated case of laryngeal diphtheria, so-called pseudomembranous croup, is not attended with either glandular swelling or fever. These clinical observations have stood the test of time and must be reckoned with. If there were no rational explanation at present we should have to wait for it, for it would not be the first time for the explanation to lag behind the observation and frequent confirmation of a fact.

For an explanation we shall have to look, if anywhere, to the anatomy of the parts. If a laryngeal pseudomembrane causes no glandular swelling and no fever, and we recognize the cause of this fact in the scantiness of the lymphatic network, we turn to the tonsil with the attempt at ascertaining whether the isolation of the morbid product on the surface or in the interior of the tonsil depends on a similar anatomical condition.

From my treatise of diphtheria published in 1880 I beg to quote the following lines:

"The tonsils are conglomerations of an indefinite number of glandular bodies, each of which has a thick capsule which is of irregular shape, and consists of connective tissue lined by mucous membrane and pavement epithelium. The connective tissue contains a number of closed follicles, each inclosing numerous lymph-corpuscles. These follicles have been considered identical with, or analogous to, the lymphatic glands; this assumption is purely problematical, since it has not been possible, thus far, to verify the existence of afferent or efferent ducts of their own. The practical deduction from this is, that the tonsils have little or no connection with the lymphatic system. The number of blood-vessels in the normal tonsil is not large, and it becomes greatly diminished when that organ has been the seat of repeated chronic inflammations. It is in these very cases of chronic inflammation and enlargement of the tonsils, with considerable hypertrophy of the connective tissue, that, for instance, diphtheritic attacks are most frequently observed, especially, of course, when a fresh irritation has given rise to inflammation and an edematous enlargement."

This statement of our knowledge of 1880 requires, as you will see, in the light of later anatomical research, some modification; one of the tonsil's functions is surely that of either preparing or storing leukocytes. In the embryonic life the thymus is the first to perform that function. Does its rôle pass to the tonsil in the born child? We should hesitate to accept that without caution, for the thymus continues to grow absolutely for several years after birth. Or are both thymus and tonsil replaced by solitary follicles and the adenoid tissue of the appendix in later life? Here again is a reason to pause. For the appendix grows smaller with increasing years, relatively, aye even absolutely. The tonsil is a type of conglomerated follicular glands, ten to twenty, according to Koelliker, eight to fifteen, according to Hodenpyl. It is perforated by crypts which unite in two or three long and deep pockets or form in rare cases a deep fissure (similar to what, according to Schmidt, is often found in certain animals).

The external adherent deep surface is covered by a fibrous capsule and outside it by the amygdalo-glossal, upper constrictor, and stylopharyngeal muscles. This fibrous capsule is of various

thickness; sometimes so disposed, according to Hodenpyl, as to send along the tonsillar blood-vessels a network of fibrous tissue which prevents them from ready contraction after having been cut. That occurrence furnishes the explanation of the occasional hemorrhages, after resections, from the tonsillar stump, though the anterior palatine artery be not touched at all. But thick or thin, coming from the fibrous membrane of the pharynx, the fibrous capsule is firm and solid.* That is why abscesses of the tonsil do not open into the maxillopharyngeal space. From it, like Hodenpyl's periarterial processes, it sends off connective tissue into and between the folds of the mucosa.

It should be added, that it also contains in its tissue thick lymphatic trunks, blood-vessels and, according to Koelliker, contracting muscular fibres emanating from the superior constrictor. Whether these lymphatic trunks can ever be active depends on the condition of the lymph circulation within the tonsil itself.

It is reasonable to conclude that as long as a tonsil is normal, not inflamed, either acutely or chronically, it will behave more or less like a normal tonsil treated on the laboratory table. That is, almost like injection material, living invading material may enter the lymph circulation, or the blood circulation, provided there is a break in a wall, but not with the same facility as injection material forced in. A surface lesion must always be supposed to exist when a living germ or a toxin is to find access. If that were not so, no human or inferior animal could exist under the clouds of microbes and toxins surrounding us. There is only one excep-

* Our textbooks are not particularly accurate. F. H. Gerrish (a textbook of anatomy by American authors, 1902, p. 730) has only this to say, that the tonsil's "external surface is attached to the upper constrictor by aveolar tissue." The textbook of anatomy, by D. J. Cunningham, New York, 1905, p. 1,036, expresses itself as follows: "The outer or attached surface of the tonsil is enclosed in a distinct fibrous capsule connected with the pharyngeal aponeurosis, outside which lie the superior constrictor of the pharynx and the internal pterygoid muscles." Gray's anatomy of 1897, p. 935—older than most references quoted by me—when speaking of the lymph circulation of the tonsils, says: "Surrounding each follicle is a close plexus of lymphatic vessels From these plexuses the lymphatic vessels pass to the deep cervical glands in the upper part of the neck, which frequently become enlarged in affections of these organs." Cunningham says only: "The lymphatics are extremely numerous and pass down to join some of the submaxillary lymphatic glands near the angle of the jaw." Retterer, Hodenpyl, Labbé and Poirier are not quoted.

tion. Stoehr has shown small gaps between the normal epithelia of the surface of the tonsil. It looks rational, therefore, to admit that when there is merely a surface lesion, though ever so slight, there may be an invasion into the tonsil. When, however, the vessels are exposed to pressure by newly formed cells or tissue, there is no invasion, certainly not beyond the tonsil itself, no ganglionic swelling, no toxic poisoning.

It is even rational to believe that now and then, when tonsils, or, what is more common, a single follicle, becomes inflamed, the very venous obstruction will exert the bactericidal influence of the stagnating blood serum.

The lymph vessels of the pharynx come from two sources, from the mucous membrane and from the muscles. The muscular network is of little importance. Even Sappey did not inject it except in cattle and the horse. The mucous network is immediately under the epithelium of the surface; and is mostly developed in the pharyngolaryngeal grooves and on the posterior wall of the larynx.*

On the level of the palatine tonsil—that is, not on the tonsil itself—the mucous network of the lymph current presents an unusual richness and its walls are very thin. Near the esophagus there are few lymph vessels.

Collecting trunks there are three: First, upper; second, middle; third, lower. The first and third do not concern us to-day. The second comes not so much from the tonsils as from the mucous membrane of the adjacent parts. They perforate the muscular tunic just above the big horn of the hyoid bone. Its ganglia are just below the posterior belly of the digastric muscle.

The second volume, p. 1297, of Poirier and Charpy's Anatomy, treating of the tonsil, says nothing of the lymph vessels and its efferent trunks of the tonsils, but the fourth volume does. I refer to p. 92. The lymph vessels of the tonsils originate, first, in the cul-de-sac descending from the surface of the crypts; second, from the ringform nets situated around the follicles; third, from the interfollicular tissue. They all form a network which finally, according to Poirier, but not to many others, passes into the fibrous capsule, where they swell into long trunks containing

* I mean to add here, not on the vocal cords; that explains, as I have done these more than thirty years, the absence of constitutional symptoms in purely laryngeal diphtheria; that is, uncomplicated "membranous croup."

valves and ganglionic enlargements and at last, together with the lymph ducts of the base of the tongue, end in the submaxillary ganglia near the angle of the jaw and near the hyoid bone. *There are, however* (Labbé), *no lymphatic sinuses around the tonsil,* such as you see around ganglia in other parts of the body.

Now comes a very important statement: Retterer (1886) concluded from his injections of gelatin and silver nitrate, that the lymph net occupies the whole follicular mass of the tonsils, and constitutes in these organs a system of closed canals which *do not open into* the connective reticulum either by open stomata or by extensive outrunners. This is confirmed by Hodenpyl, though denied by other observers.

A. Most (*Arch. f. Anat. u Entw. gesch.,* 1901, p. 86), one of the latest investigators, had the following experience: While injecting the lymph vessels of the lateral pharyngeal wall, the effect was governed by the exact location. "When an injection was made near the pharyngeal tube orifice, it would run in both directions, both backwards and forwards; when in the region of the tonsil, the injected material would run off to the anterior parts of the fauces only." This suggests limited or absent absorption posteriorly. Thus, even near the tonsil, not to speak of the tonsil itself, there is less lymph circulation than posteriorly. "The lymph vessels of the tonsils and of the neighboring mucous membranes, and of the anterior and posterior palatine arches, gather in three or five efferent trunks. They finally arrive at glands which lie laterally of the jugular vein and near or below the lateral belly of the digastric muscle. One lymph body is located in the angle formed by the anterior facial and the internal jugular." Thus, under very favorable circumstances, there must be a possibility of the contents of the lymph circulation getting away from the tonsil.

One of the very latest writers, perhaps the latest, is George Bacon Wood. He published an essay on the lymphatic drainage of the faucial tonsils in the *American Journal of the Medical Sciences,* August, 1905. Wishing to do justice to my subject, I quote from him largely, for it must be supposed that before writing a monographic essay he made himself perfectly acquainted with the literature of the subject. "It has been proven," he says, "by several very thorough and capable investigators that foreign bodies in the crypts can pass through the epithelium into the interfol-

licular tissue. This interfollicular tissue consists of a fine connective tissue reticulum interspersed with numerous lymphoid cells. These pass into the lymph spaces which terminate in the trabeculæ of the tonsil and which in their turn empty into the efferent lymphatics of the tonsil." Thus the author thinks a current is established into which the said foreign bodies are introduced. He also reports that he forced injection material by gentle massage (p. 220) into the efferent lymph ducts and a few of the lymph bodies of the neck and suggests that bacteria may get into the body by their own locomotion or by their growth. And he adds verbally: "I have not been able to find any reliable published data concerning the glands that receive the efferent lymphatics of the tonsils." And on p. 127 he has what follows: "I understand thoroughly that in advancing this idea, I am open to criticism for theorizing about a scientific subject. It seems to me, however, that the above is a probable explanation of the mechanism of tonsillar absorption and helps to substantiate the theory that the faucial tonsil is more important as a source of infection than is the lymphoid tissue on the lateral folds and posterior wall of the pharynx."

In all of this I think he is open to exactly the criticism he means to disarm. What he should set out to demonstrate he has not demonstrated.

One of the glands, under the sternocleidomastoid where it is crossed by the digastric muscle, he claims as tonsillar. His injections were not equally successful. It receives lymph from the lymph bodies of the throat also. Finally, our author says (p. 224): "The most important infections of the cervical lymphatics originate through the tonsil, and because of the importance of the courageously postulates as an anatomical fact what he proposes tonsil in the origin of the infections, I feel that the term tonsillar is a most appropriate one." He takes things too easy. What he sets out to prove he simply postulates. He takes the theory of tonsillar invasion for granted, wisely concludes there must be a sufficient number and calibre of afferent and efferent vessels, and as a theory.

A number of facts should be considered in connection with the structure and the absorbing and transmitting power of the tonsil. Its surface epithelium is, according to Stoehr, not uniform, and may admit foreign material even without a lesion. Thus an invasion into the normal tonsil is possible.

A catarrh, with destruction of epithelium, may add to that possibility.

The lymph current near the tonsil is less active than that of the pharynx at some distance. There are, moreover (Labbé), no lymphatic sinuses round the tonsil.

There is a system of closed canals *in the follicles* which *do not open* into the connective reticulum. This observation of Retterer is confirmed by Hodenpyl.

The external deep surface of the tonsil is covered by a firm fibrous capsule. Still, according to many, there is a lymph communication between the tonsil and at least one gland situated near and below the crossing of the sternocleidomastoid and digastric muscles.

Exposure, and many diseases, change the surface and the structure of the tonsil.

Every new inflammation changes them. New cell infiltration and cicatricial tissue renders absorption less possible. Blood and lymph vessels are compressed and undergo atrophy. That is why in advanced life the tonsil gets harder and smaller, and infections become less. No tonsil remains long in its original condition; and the same deleterious influences will no longer prove effective.

Laboratory injections into the normal tonsillar structure are made under pressure, and prove nothing, or very litttle, for the unembarrassed organ when *in situ,* and surely not under pathological conditions. Thus, while the normal infantile tonsil which has not suffered from previous exposure or disease may or must be believed to permit *some* invasion into the tonsillar structure, and sometimes even beyond it, microbic invasion through the tonsil is not predominant over that which takes place through the lymph apparatus of the pharynx.

That agrees perfectly with the clinical observation of local toxic affections of the oral cavity, according to which membranous affections limited to the tonsil cause no, or little, adenitis or constitutional symptoms.

MODIFIED WET NURSING.

BY THOMPSON S. WESTCOTT, M.D.,
Associate in Diseases of Children, University of Pennsylvania, Philadelphia.

During the past fifteen years, coincident with the remarkable advances that have been made in all branches of medical science, the empiric art of artificial infant feeding has been revolutionized by the labors of Rotch and many who have followed in his footsteps, and we can now feel fairly satisfied that its principles have been reduced to something like a scientific basis. The difficult problems of feeding in abnormal cases have been met and solved, and our medical schools are now sending out their graduates to all parts of the world well equipped in the theoretical knowledge of how to fit the food to the varying and often idiosyncratic needs of the infant.

There is danger, however, that in our self-satisfied contemplation of our own skill in the manipulation of feeding mixtures, and our brilliant successes in correcting the errors of our professional brethren, we may lose sight of a feeding specialist whose claims to priority cannot be disputed, and whose method of modification has defied all our efforts to furnish a substitute "just as good."

The family doctor of twenty or thirty years ago, when confronted with the problem of a hungry infant and a failing maternal breast, did not hesitate long in securing a wet nurse and starting his little patient upon a satisfactory modification prepared in the laboratory of her ample bosom.

It is an unfortunate though probably natural sequel of our advance in feeding methods, that the demand for the wet nurse has steadily decreased, and coincident with this the available sources of supply have so diminished that at the present time it is very difficult to secure a suitable woman who is willing to become a foster mother to another woman's infant. I do not know how this may be in other communities; but in Philadelphia and its surrounding territory it has become almost impossible to

secure a wet nurse at any price, and we have at length been driven to import them from the more prolific centres of foreign population, such as New York. One of the largest maternity homes in the city, from which it was once an easy matter to get a wet nurse, has within the past few years adopted the system of retaining available mothers to foster-nurse their foundlings; and even in the other institutions where unmarried mothers are cared for, the utilization of their nursing abilities as a means of livelihood does not offer the attractions that it once did. Add to this the growing independence of the respectable class of mothers, who once could be induced to assume positions as foster mother in private families, and we have an explanation of the decadence of this one-time lucrative source of employment.

As a matter of fact, the passing of the wet nurse, as we have known her often to our sorrow, would not be a source of poignant regret if we were able to secure one when her help was most necessary.

We have all met, and are still meeting, cases of malnutrition in infants, in which the peculiar adaptability of human milk at once suggests it as the quickest and most certain means of restoring a starving baby to health. In many instances the natural repugnance of the mother is an insurmountable obstacle; and the domestic difficulties attendant upon the introduction of a stranger into the household are too well known among the laity to warrant the physician's insistance, especially when he is forced to admit that in all probability he can finally accomplish satisfactory results by modern methods. A certain class of cases, however, is occasionally encountered in which nature has been so outraged by unskillful methods of artificial feeding that natural wet nursing or a modification of it seems to be the only resort if the infant's life is to be preserved. It is to this class of cases that I wish to direct attention and to elaborate somewhat the method of what may be properly termed modified wet nursing. I do not present this with any claim of originality, for I have no doubt that many members of this Society have been occasionally driven by circumstances to adopt some such expedient; but as I am unable to find any definite mention of it in the literature, it will serve as a stimulus to discussion.

By the term modified wet nursing I would indicate a method of feeding in which human milk is to be used in conjunction with

artificial food, not in the form of two or three full nursings separated by other feedings of artificial food, as in gradual weaning, but as an integral and very important part of a bottle mixture containing a cow's milk modification or other food ingredient. The necessity for some such method of administration may lie either (1) in the profound prostration and physical weakness of the baby, rendering active efforts at nursing impossible; or (2) in the absolute refusal of the infant to take the nipple, as so frequently happens when the child has already been weaned, and has become accustomed to the less laborious nursing from the bottle; or (3) when a wet nurse cannot be procured, but some nursing woman can be found, who, while residing at her own home, is willing to supply by pumping a portion of her daily yield of milk. I need not dilate here upon the vital qualities of human milk. Even a few ounces of it administered to an artificially fed infant recovering from an exhaustive illness, such as a severe enterocolitis, acts as a strong vital tonic and stimulant, bringing color to the cheek, brightness to the eye, and restful, satisfied slumber, such as no artificial food that could be tolerated at the time could possibly produce. I have repeatedly made use of temporary wet nursing as a most helpful aid in the convalescence from summer diarrheas in very young infants.

Unfortunately, as I have indicated, an available nurse often cannot be found, and thus in a number of cases I have been compelled to beg or buy a few ounces at a time from some friend of the mother's, or some respectable woman, who, while unwilling to entirely deprive her own infant of its rightful nourishment, could spare a portion of her supply to tide us over a critical period. More than once I have been able to secure small quantities of milk from two women at the same time, and in one case, the history of which I shall presently sketch, for three or four days I had three women contributing their mite of milk to make up our daily supply.

When human milk is to be used in this way it should be collected and transported with all the care that is given to the collection of milk from a dairy herd of cows. As a matter of fact, the danger of contamination of human milk thus treated is decidedly less than in the case of the dairy product. There is no exposure to stable dust and filth, no dirty udder or uncleanly milker to be reckoned with; and if the milk is drawn from a cleansed nipple,

with a sterilized pump, poured at once into a sterile bottle and kept upon ice until used, it should be nearly as free from adventitious bacterial contamination as the milk sucked directly from the breast by the nursing infant.

When a wet nurse can reside in the infant's home and can devote herself entirely to its needs, she should be properly instructed in the method of collecting her milk and encouraged to use the pump at regular intervals depending upon the abundance of her supply. If she has a superabundance, her own infant may be allowed to be with her, provided she is able to furnish by pumping an adequate amount for the foster child. As we cannot expect, even under the best conditions, to obtain by pumping as large an amount as the breast would yield to natural nursing, and never except in the earlier days as much as the foster child would need for its complete nourishment, the nurse, as a rule, may be permitted to feed her own baby, or partially nurse it with extra bottle feeding, for the purpose of keeping up the natural stimulation that direct nursing exerts upon the activity of secretion. As long as the nurse can remain under the care and observation of the physician, this is by far the better plan. But when such an arrangement is impossible, the milk from a mother in her own home may be accepted as the next best substitute.

In this plan of feeding the human milk serves simply as one ingredient of the bottle mixture. The average yield for the day should be apportioned in fairly equal amounts for each feeding, and the total quantity of the bottle made up according to the needs of the individual case.

The object of this plan of feeding is, first, to secure a food which can be best appropriated at once by the starving organism of the infant, and then to continue this until, by gradual increase, the artificial ingredients of the bottle can be fitted to the increasing demands of the infant, so that the human milk can be dispensed with altogether and the thriving infant allowed to proceed upon a satisfactory artificial food.

The urgent need and satisfactory employment of modified wet nursing can best be illustrated by the brief history of a case which came under my care last summer. On May 27, 1905, I was called to see Ruth K., born March 24th. Her weight at birth had been 5 pounds, and she had been nursed for only a few weeks. She had then been fed artificially upon the usual variety of foods

woman. She says she never had any serious illness, although all her life she lacked in endurance.

The boy took sick on the first day of September, 1905. At the dinner table he did not seem quite himself. He appeared exhausted; his face became pale; he was listless, and had to be carried to bed. Although this was quite unusual, nevertheless the mother attributed it to hard play. The next morning he vomited several times, but did not create the impression of being seriously ill. The pulse was 120 and the temperature 101° F. He was drowsy, though he could be readily aroused. The mother thought he did not act differently from many another child suffering from fever. On this day the parents were invited to a friend's farm in the country. Notwithstanding the little fellow's indisposition, they took him along. He continued to be drowsy, refused food for the most part, and vomited occasionally. This was the third day of his illness. In the evening the boy did not seem any better and the parents returned with him to the city. He acted excessively nervous, was very irritable and fretful, and it was noticed by the mother that the left side of the face was slightly drawn and a slight tremor appeared in the right hand. His condition was not considered worse on the night of arrival home, since he slept nearly all night. On Monday at five o'clock P.M. he seemed decidedly worse. His temperature was 104° F.; his pulse was very rapid. He lay in bed in dorsal position, unconscious, though he gave an occasional cry. There were involuntary passages from the bowels and the bladder. On Tuesday (the fifth day) he was unable to speak and was stuporous. He vomited occasionally, and threw himself about. At this time it was observed that he had a paresis on the left side of the face, and it was suspected that the right arm and leg were paralyzed. He lay in this condition of stupor until Thursday (the seventh day), when he awakened for an hour. On Friday he became brighter, taking notice of some figures on the bedspread and calling attention to same. From this day on he was without fever. His recovery was gradual. He shortly regained his speech, and in three weeks more was able to walk. The mother called attention to the fact that, although improved and up and about, he nevertheless remained extremely nervous. He became greatly excited in the presence of a company of people, or when taken downtown among large crowds. The slightest physical effort seemed to prostrate him.

I saw him for the first time on the fourth day of his illness, and noted a well-developed, well-nourished boy in a state of unconsciousness and with a considerable fever, varying from 102° F. to 104° F. The examination of the head and scalp was negative. Inspection of the ears by a specialist showed them to be normal. The pupils were regular, reacted readily to light and accommodation. The examination of the eye grounds proved negative. The left half of the face was drawn slightly. The tendon reflexes of the arm and leg on the right side were somewhat more brisk than those on the left. The urine was excessively acid and showed slight traces of albumin.

In the course of the disease the little fellow showed progressive improvement. There is now a very evident paralysis of the left half of the face. If he smiles or attempts to whistle, or even to speak, left facial paresis is unmistakable. The hand and arm on the right side have recovered to a very considerable degree. There is a slight flexion of the fingers, though he uses his hand with great facility. There is some limp when he walks, though there seems to be very little spasticity of the right leg. He sometimes complains of a pain in the right foot and hand, probably the socalled posthemiplegic pain. He is a bright little fellow, quick and nervous in his actions, talks about almost every subject in a very intelligent way. He makes the impression of being mentally normal.

This case presented such difficulties in diagnosis which confront one in the incipient stages of this disease. During the first forty-eight hours one would have thought of some acute infectious process; for example, an obscure pneumonia or almost any infectious disease in process of development.

The case is of additional interest in that it shows undoubted evidence of a crossed paralysis with the lesion located in the pons, and belonging to that variety already referred to as polioencephalitis inferior.

Etiology.—The causes of acute non-suppurative encephalitis may be summed up briefly in the words of infection and intoxication. Acute alcoholism possibly plays a rôle in adult and older children. In one of Wernicke's cases, poisoning with sulphuric acid was suspected. In other cases, nicotine has been thought of as a factor. The infective source is the most frequent of all in the encephalitis of children. It is well known that an encephalitis may develop during the course of a cerebrospinal meningitis.

In nearly a third of all the cases of hemiplegia in young life, it has followed one of the acute infectious diseases, occurring either at the height or the defervescence of the disease (Freud). There is probably no infectious disease known which has not been reported at one time or another as having been followed by cerebral hemiplegia. Measles and scarlet fever, according to Freud, head the list. Next in order come smallpox, German measles, whooping-cough, diphtheria, croup, follicular tonsilitis, morbus maculosis, dysentery, pneumonia, typhoid fever, mumps, intermittent fever, chorea with endocarditis; even vaccination is said to have caused hemiplegia in several cases. Perhaps the best known and the best studied of all the various infections producing encephalitis is influenza. The question is not altogether settled whether the influenza bacillus itself or its toxin produces the cerebral inflammation.

Pfuhl succeeded in finding influenza bacilli in the central nervous system of cases dying of a severe nervous form of influenza. Nauwerck succeeded in finding the Pfeiffer bacillus in a case of influenzal encephalitis in the brain and in the ventricles. Cantani has injected cultures of influenza bacillus beneath the dura of rabbits, and succeeded in producing meningitis and encephalitis. He found, too, that the same result could be produced by the injection of dead cultures as well as living cultures. The inference, therefore, is that encephalitis is due to the action of a toxin produced by the bacilli on the brain substance. There is a large number of cases in which encephalitis occurs as a primary infection; that is, where no relation can be traced to antecedent disease. In the two cases which I have cited evidence of any pre-existing infectious disease is absolutely lacking.

All authors mention trauma, such as a blow on the head or a fall, as having some direct remote relation to the disease. The developmental condition of the brain, Heubner thinks, has an influence on the causation of the disease. He finds that it is much more frequent during the first and third years of life, though it does occur at almost any age during childhood. In some of the cases, syphilis is a predisposing, if not an exciting, cause by giving rise to an endarteritis, or some other form of syphilitic degeneration; such an area might also become secondarily infected. An ulcerative endocarditis may cause an embolic process infectious in character.

PATHOLOGICAL ANATOMY AND HISTOLOGY.

Any portion of the brain may be affected, though, as it has already been stated, the cortex (superior encephalitis), the basal ganglia and the region of the medulla (inferior encephalitis) are selected areas. The cerebellum is seldom affected, although Batten recently reports a case typical as to its clinical findings and verified by autopsy. The disease is usually focal in character, though the foci are multiple and disseminated. The most striking point in examining an area of inflammation is the change of color apparent in the gray substance. It is grayish-red or violet or brownish-red, or the white substance is streaked with red. Sometimes there is a uniform discoloration or the brain substance appears streaked or spotted. On section of the brain there appeared red or yellow points which have been compared to flea bites by older authors and described as being surrounded by zones of swelling and softening. When the lesion is in the cortex the neighboring pia is usually injected and may be adherent.

Brooks, in a report of a case of non-suppurative encephalitis, summarizes the pathological findings:—

"(1) Cerebrospinal meningitis, probably secondary to cerebritis, and of the 'cellular' type as described by Delafield and Pruden.

"(2) General non-septic cerebritis affecting all parts of the cerebrum, but most marked in the cortex and particularly so in that of the motor areas. The disease is characterized pathologically by perivascular round-cell infiltration, dilation of the lymph spaces and by areas of neuroglial proliferation. The changes are further identified as inflammatory by the presence of occasional plasma cells.

"(3) Degeneration of many of the ganglion cells of the cortex. From the character of the lesions of these cells it is inferred that in most cases the changes have been of recent origin. Possibly some are postmortem alterations.

"(4) Degeneration of many of the fibres arising from the large pyramidal cells of the cortex, most marked in those derived from the motor areas.

"(5) Diffuse degeneration affecting many of the fibres passing through both internal capsules.

"(6) Inflammation of the tissues of the cerebellum, of much less marked degree than in the cerebrum, but apparently of the

same character and accompanied by similar, but much less extreme, changes in the cells and fibres.

"(7) Degeneration of many of the descending fibres of the pons and medulla.

"(8) Degeneration of the chief descending tracts of the spinal cord with degeneration of occasional fibres in ascending tracts, possibly aberrant descending fibres.

"(9) Slight, probably secondary cytoplasmic degeneration of the ganglion cells of the anterior horns of the spinal cord."

The results of acute encephalitis may terminate in local necrosis or formation of scar tissue. Glia cells are proliferated and connective tissue formed. This results in a local induration, referred to by the older writers as brain sclerosis. The process may be very extensive and lead to atrophy of a part or even an entire cerebral hemisphere. In other organs, lesions are sometimes found as the result of the general infection. The spleen is at times enlarged; hemorrhages occur into intestines and mesentery, while a parenchymatous degeneration of myocardium and acute nephritis may be present.

SYMPTOMATOLOGY.

Strumpell, in his "Lehrbuch für Specielle Pathologie und Therapie, 1893," speaks of acute encephalitis as occurring in children between one and four. The disease is marked by sudden onset, with feeling of illness and fever. Shortly nausea and vomiting, or general cerebral disturbance in the form of coma and convulsions, may occur. This condition may last one or two days, or it may last one to three weeks. The children may recover from the disease gradually, but paralysis in some form will remain. After the paralysis has existed a while, examination usually shows that both extremities of one side are affected, the arm more than the leg. There are no sensory disturbances. The growth is to a certain extent retarded. The muscles are somewhat atrophied. There is no reaction of degeneration, but almost always more or less muscular contraction. Variations from this course sometimes occur. Children occasionally die suddenly before the diagnosis can be positively made. Chronic forms sometimes occur. In these cases, the initial symptoms may last one or two months. Convulsions occur frequently, resulting from the new inflammatory areas. Strumpell believes this disease is anal-

ogous to acute poliomyelitis; the initial symptoms are so similar that in a given case it might be difficult to decide whether a cerebral or spinal paralysis would ensue. In another group of cases, which belongs more particularly to the inferior polioencephalitis group, the acute infectious diseases very often precede the onset of the encephalitis. One of Leyden's cases of this type is of interest. A fifteen year old boy fell suddenly ill with headache, nausea and dizziness, and on the third day these symptoms were augmented by fever, vomiting, difficulty in swallowing; the symptoms progressed, and on the sixth day of his illness, in addition to the headache and dizziness, he developed rigidity of the neck, ataxia, paralysis of both facial nerves, paralysis of the hypoglossal, of the soft palate and vocal cords. He also had a mild fever. His pulse was slow; there was no involvement of the eye muscles at first; later on he showed paralysis of the abducens. He died on the eleventh day of his sickness, and the autopsy showed a pneumonia with an encephalitis in the region of the medulla.

Another case of encephalitis located in the pons, reported by Oppenheim, was that of a twelve-year-old girl, who suffered from a swollen face, with pain on the left side. She also suffered from headache. Very soon she became mentally dull. There was no fever; there were no changes in the eye grounds. Some time later, however, the left facial nerve became entirely paralyzed, with a hemiparesis and incoördination of both arms, though most marked in the left arm. The tendon reflexes became increased; the soft palate was paretic; articulation was difficult. All of these symptoms pointed to a focal lesion involving the pons in the region of the left facial nerve, and extending so as to involve the sensory and left pyramidal tracts.

In the majority of cases the disease begins in the midst of perfect health, like acute infection, and in the manner already referred to. Convulsions may occur; they may be of the Jacksonian type, or they may be general. The vomiting may be persistent, and the child remain in a febrile, stuporous, or comatose condition for several days. The temperature is variable; the coma rarely complete. At first sight, one is inclined to think of meningitis or perhaps some other acute infectious disease which is in process of development, with decided nervous symptoms; but in a few days the fever declines; the child becomes more quiet, and thus, on the fourth or fifth or perhaps the tenth day of the disease, the paralysis becomes evident. There may be an arm in-

volved, a complete hemiplegia, or a crossed paralysis due to a lesion in the pons, as has already been referred to.

At the very outset the reflexes may be diminished, and a generalized paralysis of all of the extremities seems to be present. In several days, however, the unaffected extremities begin to regain their motor function, and later on the child begins to move to some extent the affected arm or leg. At this time the reflexes on the involved side are usually increased, and the Babinski toe sign becomes manifest.

In addition to the symptoms already enumerated on the part of the nervous system may be mentioned the nutrition of the skin, which is affected. It is likely to appear blue on account of capillary and venous stasis in the hand and foot of the affected side. Such an extremity is cyanotic; sometimes slightly edematous; and not infrequently cooler than the limb of the other side. The skin is dry; the sweat glands less active on the affected side. Very often and particularly in the milder cases, the intellect of the child remains practically unimpaired. At other times, where the lesion is extensive, more or less permanent mental deficiency supervenes, even to the production of idiocy. Such a disturbance of motor speech may persist in varying degree. In many cases epileptic convulsions develop from which recovery is impossible.

Schultze first called attention to the fact that deaf-mutism may result from acute encephalitis. He cites the following case:—A two and one-half year old child, healthy, received a blow on the head. The same evening it cried for several hours, and on the following night had convulsions with slight fever. For two days thereafter it was stupid, restless and refused food. On the fourth day, showed some improvement. Ten days after the beginning of the disease it was found that the child was deaf; also that it had slight strabismus; no other paralysis existed. The deafness continued, although the middle ear was normal.

In some of the milder cases, the paralytic symptoms may entirely disappear, and Medin reports a recovery without permanent paralysis. Comby also reports a case in a seven year old girl cured without a trace of paralysis.

Complete recoveries have been reported after slight attacks, usually slight paralysis remaining.

The terminal of the cases may be summarized as follows:—

(1) A small proportion of the cases do not survive, death occurring in the second or third week or as early as the first day.

(2) If the encephalitis occurs during acute infectious disease, death may result from cerebral conditions or from the original disease.

(3) Some cases improve within a few days, without any signs of disease after six or eight weeks. There is always a liability to hemiparesis of one side of the body or to epilepsy.

(4) The majority of cases with initial flaccidity give rise later to the spastic type of hemiplegia.

The diagnosis during the acute stage is nearly always difficult. One is inclined to think of an acute infectious disease, on the one hand, or a meningitis on the other. The diagnosis may be confused with that of a tuberculous meningitis. Spinal puncture in these cases is of but little assistance. If hemiplegia or cranial nerve paralysis occurs, followed by returning consciousness, the diagnosis is rendered comparatively easy. In short, if in the midst of absolute health, or in connection with an acute febrile disease, the child during the first years of life is seized with recurring convulsions, or vomiting, if a hemiplegia results, or if later psychic disturbances, contractures, hemichorea, epilepsy or idiocy develop, the diagnosis of encephalitis can be made with reasonable certainty.

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DISCUSSION.

DR. GRIFFITH.—I hope some day to report a case which illustrates the fact that in rare instances there may be a combination of poliomyelitis with inferior polioencephalitis. Spiller has proven by postmortem studies that this can occur. One would not expect, of course, to get the spastic symptoms seen in Dr. Abt's case.

SOME POSSIBLE ETIOLOGICAL FACTORS IN THE RECURRENT VOMITING OF CHILDREN.*

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AND

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The etiological factors concerned in the recurrent vomiting of children have always been hidden in obscurity. As long ago as 1861, Lombard accurately described the clinical picture of the condition and outlined the treatment much as we follow it to-day. But to him as well as to all who have followed since, the causative agent has been a stumbling-block.

Many different views have been held by the writers who have so clearly described the symptomatology of recurrent vomiting and from the number of accumulated histories all have agreed upon certain points. They have agreed that an underlying neurotic constitution, inherited or acquired, is practically the rule, and that the exciting cause is often found in fright or excitement, great fatigue, anger, sudden exposure to cold and rarely to blows upon the abdomen, all of these believed to act upon or through the nervous system. The negative side is especially worthy of emphasis. They have agreed that the attacks are not directly attributable to errors in diet.

A decided advance was made when Edsall, Pierson and Marfan showed that in the attacks acetone, diacetic acid and β-oxybutyric acid are excreted in the urine in large amount.

Acquainted with these facts, two years and a half ago Dr. Richards and I began to study this question of the etiology of these attacks, making use of material which Dr. Holt most generously placed at our disposal. Three children suffering from these attacks I have had constantly under observation

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* The analyses and experiments mentioned in this paper were conducted in the Laboratories of Pharmacology and Physiological Chemistry of Columbia University, at the College of Physicians and Surgeons, New York.

since then and have been exceedingly fortunate in having seen at least a dozen others. From a study of the urine of these cases we hoped to obtain an idea of the metabolic changes which accompany the attacks, in the belief that if this were possible we might be able, by suitable means, to reproduce the symptoms in experiments on animals.

In the first place, we have confirmed the often-repeated observation of the presence of acetone and diacetic acid. In some cases we have found β-oxybutyric acid to be present. The summary of our examination in this regard it is not necessary to give, since it is a mere confirmation of the work of others. It is sufficient to say that in the vast majority of cases of this type these substances are excreted in fairly large amounts in the urine. They are usually present before the attack of vomiting comes on, but in one case they were not found till after the vomiting had begun.

We have confirmed the observations of others in another respect, namely, that during and especially at the outset of an attack there is an increased excretion of uric acid. Some of these data are presented in the table F and more detailed reference to them will be made later. We find invariably a heavy sediment of amorphous urates, which fails to occur when the vomiting ceases.

We present in tables J. S. and McA. data relating to the excretion of sulphur. It is seen in both that the excretion of sulphuric acid diminishes while that of the neutral sulphur, *i.e.*, unoxidized sulphur, increases.

TABLE F.—MILD ATTACK. VOMITING FOR FOURTEEN HOURS.

Day.	Total N. Grams.	NH_3.	Uric Acid. Grams.	Indican.	S as SO_3.	Neutral. S.
2	10.064	1.54	0.61	++		
3	6.213	0.78	0.41	++++		
4	5.477	0.55	0.26	+++		
5	5.572	0.35	0.14	++		
6	4.892	0.20	0.19	++		
7	5.733	0.45	0.20	+		
8	6.160	0.33	0.21	++		
9	5.754	0.37	0.19	+++		
x*	10.416	0.29	0.10	++	0.52	0
y	7.168	0.20	0.09	++	0.50	0.06

* x and y are two normal days about 2 weeks later.

TABLE J. S.

	Total N. Grams.	Total SO_3 Grams.	Neutral Sulphur. Grams.	Indican.	Lactic Acid.
First day of attack	6.90	0.45	0.09	Strong reaction.	Present.
Normal day after attack	9.90	0.50	0.05	Strong reaction.	Not present.

TABLE McA.

Day.	Total S. Grams.	Sulphur as SO_3. Grams.	Neutral Sulphur. Grams.	Total N. Grams.
1	0.30	0.20	0.10	4.68
2	0.42	0.27	0.15	5.21
3	0.44	0.35	0.09	5.04
4	0.40	0.28	0.12	5.94
5	0.44	0.35	0.09	6.34
6	0.36	0.29	0.07	4.94
7	0.49	0.36	0.14	6.03
8	0.42	0.35	0.07	6.05
9	0.47	0.41	0.06	6.28
10	0.65	0.57	0.07	9.34

In J. S., also, the only specimen we have tested in this regard, lactic acid was observed to occur at the onset of the attack, and was not found after recovery had ensued.

Our results on indican excretion agree with those of other observers in showing that the quantity of indican is increased in a high degree before and in the first days of the attack. It may continue to be excreted in large amounts throughout the attack and may be detected in abnormal amounts even after recovery.

A brief consideration of the possible significance of these urinary changes will indicate the ground on which our experimental work was based.

It is now the general belief that fats yield energy to the body as the result of a series of oxidative transformations, the final products being CO_2 and H_2O. Under perfectly normal conditions this transformation is a complete one, *i.e.,* only CO_2 and H_2O are excreted and none of the intermediate products appear in the excreta. Among these intermediate products of the combustion of fats are β-oxybutyric and diacetic acids.

The recent researches of Weintraud, Rosenfeld, Hirschfeld

and others have shown, however, that the combustion of fats is not complete unless at the same time there is an adequate oxidation of carbohydrate. The processes by which fat is burned are so dependent on the simultaneous combustion of carbohydrate that, if for any reason carbohydrate fails to be burned in sufficient degree, the intermediate products of fat metabolism appear in the urine, *i.e.*, diacetic, β-oxybutyric acids and acetone. Carbohydrate may fail to be burned in sufficient amounts for either or both of two causes. It may not be present in sufficient quantity in the diet, or the body cells may have lost in a greater or less degree their power of oxidation of carbohydrate. For the former reason, the acetone bodies are excreted in starvation; for the latter reason, they make their appearance in diabetes.

If we should assume that this diminution in carbohydrate combustion is the *sole* cause for the elimination of acetone bodies in these cases of recurrent vomiting, then we must decide on what factor the limited carbohydrate combustion depends.

One consideration leads us to believe that it does not arise as a result of carbohydrate starvation—namely, that the interval of time in many cases which exists between an apparently normal condition of the child and the onset of vomiting with the appearance of these substances in the urine is no longer than elapses between two consecutive times of feeding. In such cases it is obvious that the factor of starvation cannot be the essential factor. It seems more logical to assume that it is mainly due to a diminished power of oxidation of the body cells.

In any case, in these attacks the acetone bodies are probably excreted as the result of lessened oxidation of fat, brought about either indirectly as the result of lessened sugar combustion, or by a direct influence of some unknown nature on fat combustion.

Uric acid is one representative in the urine of the metabolism of nuclear compounds. Its excretion may be markedly increased by the addition of nuclear substance or of nuclein derivatives to the diet. In these cases, however, its increase cannot be due to such a cause as this, for the composition of the diet is either unchanged or is very much diminished. It must arise, therefore, as a product of the decomposition of nucleoproteids of the body, *i.e.*, it is largely *endogenous* uric acid.

The nucleoproteids are decomposed in metabolism with the formation of—

Adenin	$C_5H_5N_5$
Guanin	$C_5H_5N_5O$
Hypoxanthin	$C_5H_4N_4O$
Xanthin	$C_5H_4N_4O_2$

The enzymes guanase and adenase transform guanin and adenin into xanthin and hypoxanthin respectively:—

$$C_5H_5N_5O + H_2O = C_5H_4N_4O_2 + NH_3$$
$$C_5H_5N_5 + H_2O = C_5H_4N_4O + NH_3$$

Hypoxanthin and xanthin are then transformed into uric acid by oxidation brought about by the influence of an enzyme, oxidase;

$$C_5H_4N_4O + O_2 = C_5H_4N_4O_3$$
$$C_5H_4N_4O_2 + O = C_5H_4N_4O_3$$

Uric acid.

Not all, however, of the uric acid thus formed is eliminated as such. Under the influence of a "uricolytic" ferment, recently discovered, uric acid is further broken down by oxidation, so that in man only about one-half of the uric acid *formed* is eliminated in the urine as such.

To return to these cases, then, we cannot explain the increased elimination of this substance by change in the composition of the diet. It could, therefore, be due to an increased formation or to a decreased oxidation of uric acid. That it is due to decreased oxidation is suggested by the figures for total nitrogen.

In the table in which the uric acid estimations of F are given, it is seen that while the nitrogen values of days three and four, or four and five, are nearly constant, there is a very marked decrease in uric acid. If this decrease in uric acid, which, as the other figures show, is a decrease toward the normal and doubtless has the same significance in a reversed sense as the initial increase in uric acid, were due to a lessened metabolic decomposition of nuclear compounds a decided change in the nitrogen elimination would probably accompany it. That is, at the same time that the purin bases are split off from the nucleoproteids, metabolism of the nitrogenous portion of the nucleoproteids takes place and increased elimination of nitrogen would result. Since the proportion of purin bodies which can be obtained from any single nucleoproteid is small (1 per cent.) and since the amount of

nitrogen is large (16 per cent.) it is obvious that an increase in uric acid (which represents increase in nuclein metabolism) must be accompanied by a far greater increase in total nitrogen. These considerations have led us to the conclusion that the origin of the increase in uric acid in these cases, not being due to change in diet, or to increased nuclein metabolism, must be due to decreased destruction. Since the destruction of uric acid takes place as a process of oxidation, we may conclude that oxidation of uric acid at the outset and in the first periods of the seizures we are studying, is interfered with.

Additional evidence of interference with oxidation is shown by the elimination of sulphur of the urine. Normally, about 90 per cent. of the total sulphur is excreted in the form of sulphuric acid, *i.e.*, oxidized sulphur. Most of the SO_3 arises from the oxidation of the sulphur-containing radicals in the proteid of the diet. Most of the unoxidized sulphur arises from the metabolism of the sulphur-containing proteids of the body cells. A decrease in the diet would therefore, by lessening the SO_3 in a greater degree than the neutral sulphur, increase the ratio. For this reason it is found that the ratio of neutral sulphur to sulphuric acid sulphur in starvation increases. But it is equally obvious that in such a condition the total sulphur will diminish. In our cases, especially J. S., this is not found to be the case. The total sulphur of the two days analyzed remains the same, while the neutral sulphur rises and the oxidized sulphur falls. We believe this change to be due not to starvation, but to an interference with the processes by which sulphur is oxidized to SO_3.

Still further and more convincing than all is the observation of the occurrence of lactic acid. Lusk has shown very recently that in the combustion of dextrose, lactic acid is formed as an intermediate product. It had previously been shown that lactic acid introduced into the body is completely oxidized, *i.e.*, none of it reappears in the urine. In conditions of incomplete oxidation, however, lactic acid appears in the urine spontaneously and if injected is excreted as such. It is therefore a decidedly abnormal constituent of the urine and as much as any other constituent may be looked upon as an indication of decreased oxidation.

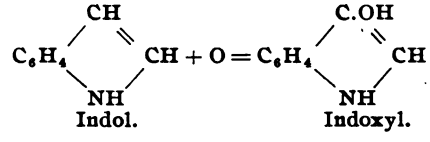
To sum up these conclusions, we believe that the figures we have quoted and the considerations which we have offered justify the belief that at the outset of the attacks there is an interference

with certain metabolic processes in which oxidation plays a part; that as the attack wears off the urinary signs of this interference wear off.

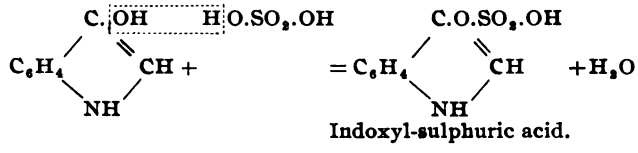
It has been stated that in the urines of these cases we have confirmed the observation that abnormally large amounts of indican are excreted before the attack. This indicates, of course, an increase of putrefactive processes in the intestine, with an increase not only of indol, but also of skatol, phenols, etc. We have given our reasons for the belief that at the same time there is a lessened power of oxidation in the body.

The question at once arose of the possibility of a relationship between these two factors.

Urinary indican results as a synthetic product of the oxidation of indol:



Indoxyl is converted into indican by combination with sulphuric acid.



The same changes take place in the excretion of skatol, which is simply methyl indol.

Indol and skatol then require oxidation before transformation into the final form in which we find them in the urine.

Phenol, however, combines directly with sulphuric acid, forming phenol-sulphuric acid. It has been argued by some competent observers that this power of the cells of effecting the combination is dependent upon simultaneous oxidation; that influences which diminish the power of oxidation lessen power of phenol synthesis.

Comparatively few data regarding the toxicity of indol and skatol exist. Large amounts (18 grams) have been administered to healthy animals with the production of no symptoms. The matter is summed up in Kunkel's Toxicologie with the statement

that from the experimental evidence at our disposal we must conclude that indol is non-toxic. This indicates in a striking manner the adequacy of the organism for transforming substances of this type.

If, however, the ability of the organism to effect this oxidative transformation is diminished it is possible that any toxicity which these substances possess will be exhibited. Our animal experiments are designed to throw some light on this question.

As means of diminishing oxidations we have made use of potassium cyanide and of chloroform. Potassium cyanide has been shown very definitely to diminish the ability of cells to utilize oxygen even though oxygen may be present in normal amounts in the blood. Chloroform possesses the same property in a lesser degree.

In the first series of experiments on rats, guinea pigs and rabbits, the effects of definite doses of phenol and indol in normal animals were compared with those produced by the same doses in animals previously injected with small amounts of KCN or previously subjected to chloroform anesthesia. The results are shown in the following table.

ANIMAL EXPERIMENTS.

No.	Animal	Poisons Used	Convulsions Produced		
			Onset in	Duration	Intensity
1	Guinea pigs....	KCN + Phenol	4 minutes	75 minutes	Great
		Phenol alone	4 minutes	25 minutes	Slight
2	Rats	KCN + Indol	1 hour 13 minutes	11 hours	Great. Death after 11 hrs.
		Indol alone	1 hour 8 minutes	3½ hours	Slight
3	Guinea pigs....	KCN + Indol	2 minutes	5 hours	Great
		Indol alone	8 minutes	1½ hours	Slight
4	Rats.....	$CHCl_3$ + Indol	13 minutes	8 hours	Marked
		Indol alone	6 minutes	3 hours	Marked
5	Guinea pigs....	$CHCl_3$ + Phenol	5 minutes	50 minutes	Marked
		Phenol alone	7 minutes	8 minutes	Very slight
6	Rabbits.....	KCN + Phenol	After intravenous infusion of 1 c.c.	2 hours	Marked
		Phenol alone	of 4 c.c.	45 minutes	Less marked

From these we conclude that diminution of oxidation intensifies and prolongs the poisonous effect of a moderate amount of either phenol or indol.

Our experiments on dogs afford a better idea of the nature

of the effect which indol is capable of producing when its oxidation is interfered with. We have not yet extended our studies in dogs to phenols and skatol.

In the first place, we convinced ourselves that the quantities of indol (0.25-0.5 gram) which we used in combination with the other poisons (KCN and $CHCl^3$), given alone, produced no symptoms. If, however, a dog is poisoned by repeated small doses of potassium cyanide which are sufficient to produce distinct but not fatal symptoms of cyanide poisoning, both before and after the introduction of the indol, very marked symptoms result, which cannot be attributed to the action of the cyanide alone.

In the first experiment it was difficult to attribute any of the symptoms to the effect of the indol in the hours directly succeeding the injection. There was muscular twitching and evidence of increased peristalsis of the intestine, which may have been due to this substance. But, after ten to eighteen hours, effects were evident which were not due to the cyanide alone—and which we attribute to the action of indol—a condition of stupor and semi-consciousness came on which was interrupted by periods of wild delirium, marked by frantic, uncoördinated movements and shrill screaming. The delirium came on at times spontaneously, and always followed the stimulation of rough handling or rectal irritation produced by the insertion of a thermometer. This condition lasted till the death of the animal a week after the indol was given. During this period there was marked exaggeration of the reflexes, spasticity of the hind limbs, total blindness. During the whole period, food or water was not voluntarily taken; water given by a stomach tube was sometimes retained, though there were signs of nausea. Milk, diluted milk, or beaten egg, given in the same way, was immediately vomited. Throughout the whole period there were noticed in a marked degree the typical gurgling sounds of increased intestinal peristalsis, but no feces were passed.

Two other animals treated in the same way with a proportionally larger dose of indol developed identical symptoms and died on the third day.

In two dogs, a single injection of cyanide, accompanied with a smaller dose of indol, was given. One dog was killed after thirteen days, during which time the prominent symptoms were blindness, mental depression, with some delirium, increased re-

flex excitability, enormous appetite associated with increasing emaciation, constant nausea and bloody feces.

The second dog died after a period of five weeks, in which the blindness and nausea were the most prominent symptoms. During the three days preceding death, however, he developed the mental signs described above.

If, instead of potassium cyanide, prolonged anesthesia with chloroform or artificially induced asphyxia be used as means of reducing oxidation, strictly comparable results may be obtained. It should be mentioned that in the case of chloroform the gastrointestinal symptoms seemed to be more marked than with potassium cyanide. Not only did the feces contain blood, but bloody material was vomited.

An interesting fact has been noted with regard to the excretion of indican. The doses of indol which we have used will be entirely excreted by a normal animal in the form of indican in twenty-four hours. If at the same time its oxidation is diminished, the amount in the urine becomes less and its excretion is prolonged over a longer period of time (forty-eight hours or more). This emphasizes the important clinical fact that the amount of indican in the urine depends not only upon the amount of indol formed and absorbed, but also upon the capacity of the body cells to oxidize it.

At the autopsies of the animals which have been poisoned as we have described, the results are very uniform: Marked congestion of the gastrointestinal tract, especially of the duodenum, lower part of the ileum and colon with extensive necrosis of the mucosa and even of the submucosa. There is blood in the intestinal contents. The liver showed very great degenerative change, there was excessive cerebrospinal fluid and the tissue of the brain was soft. The more acute the poisoning the more marked were the changes in the gastrointestinal tract.

That the effects which we have described are due to the indol and that they are not due to the direct effect of the poisons which we have combined with it we are convinced. In the first place, control experiments on potassium cyanide alone agree with the very extensive experience of Geppert and others, that when recovery from cyanide poisoning occurs at all it is rapid and complete, the only after effects being weakness from exhaustion. In the second place, we are able to produce the same symptoms with

the second drug, chloroform, which bears no resemblance to potassium cyanide, save in its power to affect oxidations. Thirdly, we are able to produce the symptoms by mechanically reducing oxidation, *i.e.*, by asphyxia.

It is apparent, then, that the symptoms of this form of indol poisoning may be divided into two groups, nervous and gastrointestinal. We made one experiment designed to afford an explanation of the gastrointestinal disturbance.

In one dog a section of the small intestine, 8 to 10 inches long, was severed from the main gut, its ends sewed to the abdominal wall, and an end to end anastomosis of the main gut established. A month later, when recovery was complete, the dog was poisoned with KCN and indol in the manner previously described. The urine gave no reaction for indol, but it could be detected in washings of this isolated loop of intestines. Furthermore, the odor of indol could be detected at the abdominal opening of the fistula.

This experiment indicates that if indol is circulating free in the blood, owing either to abnormally great formation or to abnormally decreased oxidation, it is excreted by the intestine rather than by the kidney.

We have proof that when the power of oxidation is diminished, indol, ordinarily non-toxic, is a most powerful poison. It is a matter of observation that children subject to attacks of recurrent vomiting exhibit evidences of increased intestinal putrefaction by an increase in the urinary indican whose precursor is indol, and often by foul stools. During the attack, constipation is the rule, which would still more increase the absorption of indol. The presence of indol in comparatively large amounts can, therefore, be assumed, and we have shown that during these attacks diminished oxidation is present.

We have evidence that the toxicity of phenol is increased, but have not sufficiently studied this nor skatol and the cresols. Since the transformations that these undergo in the organism are the same as indol and phenol, there is every reason to believe that the results will be similar. Let it be understood, however, that we do not claim that these aromatic substances are alone the cause of these attacks. It is possible that other substances, absorbed from the intestinal canal or formed in intermediary metabolism, may be also responsible.

We have not produced in animals symptoms identical to those in children and probably shall not be able to do so. The diminished oxidation in children is moderate but prolonged. Our imperfect methods allow us to produce an intense, but evanescent, diminution of oxidation in animals. As soon as this wears off, the poisons are rapidly eliminated by the kidneys; before it wears off, it is probable from the experiments previously mentioned, that the gastrointestinal tract attempts to get rid of them. This would seem to account for the gastric symptoms. It is only while under the influence of the poisons that there is diminished oxidation and the poisoning is so profound that the animals are practically anesthetized. While this was wearing off they did show gastrointestinal symptoms, vomiting, sometimes bloody, nausea and bloody stools. In children, with the moderate but prolonged diminution of oxidation, gastric symptoms can readily occur, and we have reason to believe that these substances are eliminated by the stomach, from the examination of the vomitus of F. Two samples of vomitus, clear and not bile-stained, contained a trace of indol.

These aromatic substances, as the experiments show, in large enough amounts are convulsants, and it is suggestive to read that one of Snow's patients up to the age of five years always had a convulsion before each attack and that Langmead's fatal case terminated in convulsions.

To compare the autopsy findings is unsatisfactory for the reason that few postmortem examinations on children dying from recurrent vomiting have been recorded, and those in a most meagre way. Griffith found necrotic changes in the stomach and intestines. Marcy's case was carefully examined. The stomach showed extravasated blood under the mucosa with apparent softening; this was also marked throughout the intestines. Microscopically he found almost complete necrosis of the entire mucous membrane of both the stomach and intestines. Most of the epithelial cells did not stain at all, they being represented by an indistinct granular mass; the necrosis appeared to have invaded the submucous coat. There was fatty infiltration of the liver and slight parenchymatous degeneration of the pancreas, spleen and kidney.

As mentioned above, our dogs showed very great congestion of the gastrointestinal tract with extravasations of blood. In

places the mucous membrane was very necrotic and there was enormous degeneration of the liver.

It appears to us in the light of our present knowledge that a shock, excitement, fright, anger or something of the kind, is exerted upon an unstable nervous system, unstable by inheritance or development, and also by age; for we cannot doubt that similar shocks are felt at a later time, but in the course of growth the brain and nervous system acquire a more stable equilibrium. As a result of this, in some way or other, unknown to us, a diminished power of oxidation results and the organism loses the power to detoxify substances absorbed from the intestine which have been present there in excess; these circulate in the blood, exerting their poisonous action and cannot be excreted by the kidneys because they are not brought to them in the proper form. It seems probable that they are excreted and reabsorbed by the stomach and intestine, in the light of which vomiting would appear to be eliminative and thus a protective mechanism.

Finally, the power to oxidize and detoxify these substances returns; they are rapidly eliminated, and quick improvement results.

DISCUSSION.

DR. GRIFFITH.—The paper is upon a subject in which I have felt great interest. It touches a part with which I was myself entirely unable to deal, viz., experimental work upon the pathological physiology. The impression which I got from hearing the paper read is that nearly every writer up to the time of Edsall had regarded this as a pure neurosis. I do not think that this represents quite correctly the state of affairs. Certainly, my own expression in an article published on the subject some years ago was strongly that the disease was a toxic neurosis—not a pure neurosis at all in the ordinary sense. I was not able to discover any influence exerted by fright, shock or nerve strain, and some of the cases were studied with great care as to this very point. In some instances, however, there does seem to be such an influence, and in a case observed subsequently the attacks were apparently traceable to over-exertion.

The relation of acetone to this disease has been spoken of repeatedly in medical literature. In the 2 fatal cases I reported the urine was examined by a colleague, who at once expressed the view that the child was suffering from acetone poisoning. I maintained that acetone did not produce the symptoms from which the child died, and that it was the result and not the cause

of the condition. It is unfortunate that this has been described as acetonemic vomiting by French writers, as this gives an impression which is far from representing their opinion. Marfan distinctly states that the acetone is not in his opinion the cause. Dr. Edsall's paper was the first to throw light upon the subject as regards the condition being possibly an acidosis. Although good results were obtained in some instances by the administration of bicarbonate of soda to counteract this acidity of the blood, there have been cases since then that have not been benefited by this plan at all, and we are again somewhat at sea as to the cause.

I would ask the writer of the paper not to make a distinction between Marcy's two fatal cases and mine; they were the same cases, seen by me in consultation with him.

DR. EDSALL.—I have been very much interested in this work and in the important results that Dr. Howland and Dr. Richards have obtained.

I think that Dr. Griffith is quite right in the statement that he has just made, that a considerable number of cases of recurrent vomiting respond, at best, only partially to treatment with large doses of alkalies. I am not surprised at this, for I said, when I first discussed the possible relation of acid intoxication to these cases, that acid intoxication is always a secondary condition dependent upon some preceding cause, and that controlling it is merely controlling a symptom. It is, however, sometimes the most important symptom, and controlling it in such a case is the essential part of the treatment. I have seen at least 3 cases in which the treatment with large doses of alkali seemed to exercise such absolute control over the attacks that they have for several years entirely ceased.

I have been casting about, for the last year or so, for some possible explanation of the occurrence of the acid intoxication; and, as I told Dr. Richards, in conversation with him, I had rather expected that he and Dr. Howland had perhaps been studying the condition along the lines that have recently been used in studying the acid intoxication following chloroform-anesthesia. The recent valuable work of Alonzo Taylor, for example, has shown that in a case of acid intoxication following chloroform-anesthesia autolytic processes were abnormally increased, and apparently the acid intoxication was secondary to this. This seems to me to offer possible explanation of the occasional good effects, as well as of the failures, that are seen when alkalies are used in these cases.

The work of Rowland and Hedin has indicated that autolytic processes are favorably influenced by acids and unfavorably affected by alkalies. It has, therefore, seemed to me possible that the effect of alkalies in these cases might be twofold; that is, that they would decrease any acid intoxication present, and would thereby control the symptoms due directly to the acid intoxication,

and, at the same time, by decreasing the amount of acid present, they would tend to control any excessive autolytic changes going on. In some instances, the acid intoxication might be the most important abnormality present from the symptomatic standpoint; and, in such cases, alkalies would control the symptoms. In mild cases of increased autolytic activity, this condition would likewise be controlled; but in cases in which the disturbance of autolysis had already produced severe lesions, it would be impossible, of course, by merely controlling the acid intoxication, or even by halting the increased autolysis, even if this is feasible, to control the symptoms due to the tissue destruction that has already been accomplished. This possible explanation would, I think, give an indication of the reason why alkalies appear in some cases to abort the attacks successfully, and to be, when useful at all, most useful in the early stages of the attacks. These suggestions are merely suggestions, as they have not yet been worked out; but it is possible that they may help us to understand the condition.

The results that Dr. Howland and Dr. Richards have obtained demonstrate clearly, as they say, that there is a profound disturbance of the organism at the time of the attacks; but I cannot help feeling that they can be brought quite as directly into the line of reasoning that I have suggested, as they can be explained on the basis of changes in oxidative processes alone. For example, an increase of neutral sulphur and uric acid in the urine is, of course, observed in cases in which oxidation is supposed to be disturbed. As a matter of fact, however, we say, to my mind, more about disturbed oxidation than we actually know, and it is an exceedingly complex matter to determine positively that any particular individual has decreased oxidation. On the other hand, an increase of the neutral sulphur and uric acid of the urine is known to occur in conditions in which there is an increase of tissue-destructive processes; and it may quite as well, therefore, be an indication of increased autolysis as of decreased oxidation.

Furthermore, while the experimental results in animals are highly interesting as demonstrating the possibility of severe toxic effects from indol when given in combination with other substances, I am not yet convinced that the results that have been described have any definite relation to changes in oxidation. While hydrocyanic acid does have an influence upon oxidative processes, its influence is by no means limited to this alone; it is, for example, known to have, in addition to this, a profound effect upon many ferment processes. A point of importance in this connection is the fact that the normal antidoting of indol in the body is not merely a question of oxidation to indoxyl, but is also a question of its combining with sulphuric acid to form indoxyl sulphate. The synthesis is probably quite as important as

the oxidation; and, indeed, it is not improbably more important in this as well as in other antidotal processes in the body. For example, in the detoxication of phenol in the body, a similar synthesis with sulphuric acid occurs, but without the oxidation; and again, in the detoxication of benzoic acid, the process consists simply in synthesis with glycocoll to form hippuric acid.

The relation of these facts to the experimental observations that have been reported in this connection with hydrocyanic acid is that it has been clearly shown that hydrocyanic acid interferes with the synthetic action as well as the contrary action of some ferments, both organic and inorganic; and this is clear evidence that its important effects are not limited to those exercised upon oxidative processes. It is, too, not at all clear that the experimental results reported have actually any relation to recurrent vomiting.

These apparent criticisms, however, are merely academic criticisms of the explanation that has been offered of the results obtained. The practical point in view is that these investigations have shown very remarkable and important changes in the urine, which indicate profound general disturbance of the organism of the child at the time of the attacks; and the experimental results with the cyanides and chloroform are extremely interesting.

DR. FREEMAN.—I have had recently a very pretty illustration of the relation of shock to these attacks. A child five years old had attacks at intervals of six weeks to three months, and was kept free from attacks for a year by watching and making examinations of the urine and by using on the slightest suggestion of an attack calomel and alkalies and rest. Early this spring he had a fall from a bicycle, striking the back of the head, this shock being immediately followed by a typical attack lasting three or four days and accompanied by marked acidity of the urine with diacetic acid and acetone.

DR. RICHARDS.—In regard to Dr. Edsall's remarks as to the influence of chloroform on the autolytic processes and the influence of the drugs we used, it has been shown by Herter and Wakeman that autolysis after chloroform poisoning is increased, as it is under the influence of phosphorous poisoning. These observations are the results of the estimation of hexone bases which could be derived by hydrolysis of the liver. They studied the cyanides in the same way and could obtain no such results. While the ferments may be interfered with by the cyanides the mechanism would seem to be different from that which obtains with chloroform.

We are aware that changes in the excretion of uric acid and sulphur compounds are explainable on the grounds of increased autolysis as well as of decreased oxidative changes. Lactic acid, however, is looked upon as a product of "anaerobic metabolism" of sugar. The fact that this substance was excreted at the same

time with the altered amounts of uric acid, sulphur compounds and acetone bodies, led us to the view that oxidative changes involved in the transformations of these substances and their precursors were interfered with. The possibility that this interference is the result of autolytic change in the liver is not excluded. The idea naturally occurred to us that if this interference extended to the transformations which some of the putrefactive products normally undergo these substances might then show increased toxicity. Our experiments were designed to demonstrate this general proposition, and they have shown, we think, that these substances, most notably indol, are far more toxic if given with certain other poisons. One of these, cyanide, exerts a rather specific influence upon the power of cells to utilize oxygen; the other, chloroform, possibly by increasing autolysis, disturbs metabolic processes, among which are oxidations.

As Dr. Edsall has indicated, it is not proved that the conditions which are present in the human body in recurrent vomiting are reproduced in our experiments. We cannot claim that indol or phenol are the sole, or even the main, causes of the attacks. But we believe that by experimental means we have shown somewhat more clearly than has yet been done the character of the effects which may be brought about by some of the products of intestinal putrefaction if the functions which normally accomplish their transformations are temporarily depressed; and that among these effects gastrointestinal symptoms are prominent. We also believe that the urines of the cases of recurrent vomiting which we have examined present indications of a similar interference, but of undetermined cause.

DR. JACOBI.—As to the uric acid in the urine, is it cause or effect?

DR. RICHARDS.—I think it is entirely effect.

TUBERCULOUS CERVICAL LYMPH NODES IN AN INFANT OF FOUR MONTHS.

BY L. E. LA FÉTRA, M.D.,
New York.

Simple acute adenitis is common in early infancy; but tuberculous adenitis is very unusual, in this country at least. This case is reported because of its rarity and because of certain interesting questions that arise in connection with it.

The Family History of the little patient sheds no light upon the origin of the affection. Both parents are living and well; neither has ever had a sign or symptom suspicious of tuberculosis in any part of the body. The sister of the patient, a little girl of four-and-a-half years, has been under my care since birth, as a feeding case, and has never had a prolonged cough nor any tuberculosis of the lymph nodes, bones or joints. No nurse nor any servant nor any visitor to the house since the infant's birth has had a prolonged cough. The father of the patient had several colds in the head, repeated one after another, for some weeks before and after the time of the infant's birth.

Personal History.—Allan G. N., Jr., was born October 12, 1905, at full term, the result of the second pregnancy. He was strong and healthy, weighing seven pounds and six ounces at birth. Because of scanty breast milk his nourishment from the beginning was a combination of maternal nursing and bottle feeding. The artificial food was made from the milk of a well-known dairy which has the certification of the New York County Medical Society. The milk is produced under the best conditions upon a model farm; the cattle are Jerseys and Alderneys. After a small initial loss in weight the baby began to gain nicely, weighing eight and one-half pounds when three weeks old, a gain of eighteen ounces in the first twenty-one days. The weather being fine he was sent out of doors after the second week, and all went well until he was about three weeks old, November 4th, when he developed a cold in the head, snuffling and snorting to get his breath, especially during the latter part of the night. There were no

signs in the chest, and the temperature was not over 100° F. This head cold passed off in one week and the baby was well until November 23d, when, at the age of seven weeks, he developed a second cold, this time in the head, throat, tonsils and chest. The tonsils, especially the right, showed a little exudate in the follicles, but very little redness or swelling; there were a few large râles in the chest. The temperature gradually fell from 102° F. the first day to normal on the fourth day.

Under inhalations and internal treatment these conditions were soon improved and the baby was well, save for some snuffling, for two weeks. On December 11th the baby had the third attack of "cold," this time more severe than before. There was fever up to 102° F., much prostration, dyspnea with respirations 60, and the signs of a generalized bronchitis involving the finer tubes. On the fifth day of this attack Dr. Holt saw the baby with me in consultation, and there was serious question of bronchopneumonia. It was decided, however, that the baby had only a severe bronchitis, probably a part of the general grip infection of which it was difficult to get rid. This attack lasted thirteen days, the temperature reaching normal on December 24th, and the chest signs disappearing soon afterward. On December 21st, however, when the baby was ten weeks old, there had been a rise of temperature to 101⅘° F., and at that time there appeared a slight swelling of the right side of the neck, in front of the sternomastoid muscle; this swelling consisted of three lymph nodes and remained the same size for about ten days; then it increased rapidly for a few days, but without rise of temperature over 100½° F. By January 18th it was as large as a walnut and quite hard and the node outlines were lost. Then the swelling diminished, and by January 29th the three nodes were again distinctly felt, firm, not tender and movable. For ten days they remained about stationary, perhaps a little smaller, and then the perinodular swelling reappeared and the whole mass became much larger. The outlines of the separate nodes were lost and there was indistinct fluctuation in the tense lobulated mass.

Because of the tender age of the patient, I called in consultation Dr. Charles N. Dowd, whose experience in cervical lymph node cases in children has been very extensive. He thought the condition probably subacute suppurative adenitis, and in accordance with his advice it was planned to open the abscess before the skin became badly involved. On February 17th, under ethyl

chlorid anesthesia, Dr. Dowd operated on the neck swelling. Upon reaching the lymph nodes they were found quite large, soft and hyperplastic; there was no suppuration, contrary to both our expectations, and the nodes when opened showed greyish granular material, but no distinct pockets of liquefaction. There was very little fibrous tissue about the lymph nodes. No attempt was made to do a complete extirpation of all the lymph nodes palpable in the wound; only the largest and most accessible were removed.

A few days later Dr. William C. Clarke reported the microscopic examination from the Department of Pathology of the College of Physicians and Surgeons as follows:—"The lymph nodes from the cervical region of Baby N., operated on by Dr. Dowd, February 17th, are enlarged and softened. Microscopic examination shows extensive early tuberculosis. There are many areas of degeneration and necrosis in the lobules of the nodes. These areas are surrounded by a slight amount of productive inflammation." On account of his interest in this peculiar case, Dr. Clarke showed the sections to several other workers in the laboratory, who confirmed his diagnosis.

The subsequent course and appearance of the wound have been and are typical of tuberculosis; cheesy sloughs continue to come away from the deeper nodes involved, and the skin edges have the blue gelatinous appearance which is diagnostic of this form of inflammation. The mass of the swollen glands in the neighborhood is becoming smaller, and altogether the wound is doing very well. Repeated careful examinations of the lungs, the last time on May 25th, fail to show any signs of tuberculosis, either of the lung or of the bronchial lymph nodes. The infant's digestion has been excellent all along, and his general condition is very good, considering the constant drain of the discharging wound. At the time of the operation, February 17th, his weight was twelve pounds, six ounces; on May 24th he weighed fourteen pounds, thirteen ounces, a gain of two and one-half pounds in a little over three months.*

How shall one account for the tuberculous cervical lymph nodes in this case? It is probable that the first "cold" was taken from the father or from the street dust, and that later, perhaps, through the inflamed tonsil or nasopharynx, the tubercle bacilli

* During the summer of 1906, while away in the country, the baby succumbed to what was apparently an acute enterocolitis: no necropsy was obtained. The wound had never healed.

penetrated to the lymph streams leading to the deep cervical nodes.

In connection with this case, it is of interest to consider, first, the frequency of tuberculous cervical lymph nodes in infancy; second, the source of infection in these cases; and third, the question of latent tuberculosis of the lymph nodes.

RARITY OF TUBERCULOUS CERVICAL LYMPH NODES IN INFANCY.

Although there are abundant statistics upon tuberculous lymphadenitis, there are very few authors that report cases of the disease in early infancy. The cases are grouped as "one to five years," or "one to ten years," as a rule. The affection is very common about the age of the second dentition. Billroth reports that in his experience scrofulous and tuberculous lymphadenitis occurs most frequently between eight and twenty years, but may appear at any age from one to sixty years. He says that the small glandular swellings that come under observation in children under five years old prove mostly to be acute or subacute abscesses. Fränkel made a strong distinction between scrofulous and tuberculous lymph nodes, considering the former a disease of childhood, the latter a disease occurring most frequently between fifteen and thirty years. Among 148 tabulated cases, there was only 1 of Fränkel's under five years.

Fischer, collecting 1,484 cases from various authors, found 59, or 3.9 per cent., under five years of age. Many authors with an experience of over 100 cases had seen only 1 or 2 under five years. Grünfeld, in 214 cases, not 1 under five years. In contrast to these are the reports of Manson, who had 17 under five years in 95 cases, and Gellhaus, who had 8 under five years among 60 cases. Wohlgemuth, however, among 430 cases found the ages as follows:—Up to one year, 54 cases, or 12.5 per cent.; from one to three years, 104 cases, or 24.15 per cent.; from three to five years, 46, or 10.7 per cent.; from five to ten years, 90, or 20.8 per cent.; altogether, 68 per cent. of cases under ten years. Wohlgemuth's figures are so different from all others that they demand an explanation. This is to be found in the fact that his cases are drawn from the out-patient service of the Jewish Hospital, and that these patients are mostly infants and young children living in most unsanitary conditions. As to the region of the body occupied by the affected lymph nodes, the neck was implicated in 93 per cent. of Wohlgemuth's cases. Harbitz, in his

study of latent tuberculosis, referred to later, found 10 cases in patients under one year. Among these the cervical lymph nodes were involved in 9 cases, and alone 8 times.

In this country comparatively little has been written on the subject; but what evidence there is goes to prove that in infants the condition is very unusual. Freeman reports that in 158 autopsies on tuberculous subjects at the New York Foundling Hospital the cervical lymph nodes were macroscopically involved in only 14 cases, or 9 per cent. He considers tubercular cervical lymph nodes as very rare in infants, but frequently the first lesion after the third year. Dowd writes me that my patient is the only case under one year that he has seen in a personal experience of 200 or more cases of tuberculous cervical lymph nodes and that he has had very few under the age of two years. Among 61 of his reported cases, only 3 were under three years of age.

We are, then, I think, justified in concluding that while tuberculosis of the cervical lymph nodes does occur in a small per cent. of cases in patients under three years, the great majority of these cervical swellings are not tuberculous, and that the chances against tuberculosis increase inversely with the age of the patient; *i.e.*, the younger the patient the less the probability that the swollen lymph nodes are tuberculous, and the greater the chance that they are simple inflammatory enlargements due to ordinary pyogenic cocci.

Source of Infection.—In any particular case it may be impossible during life, or even after necropsy, to determine the original site of invasion or the source of infection. Breathing and swallowing are, of course, the only possible avenues of infection, and invasion must take place in the case of the cervical lymph nodes through the mucous membrane of the nasopharynx, oropharynx, tonsils or adenoids. Tracing the infection backward from the lymph nodes along the lymph vessels that drain into the deep cervical chain, one comes upon the tonsils, the adenoids and the nasopharyngeal mucous membrane as the most likely sites of invasion. It is believed by many that the most frequent local sources of infection of the cervical lymph nodes are hypertrophied tonsils and adenoids. Either the tonsils and adenoids may be tuberculous, or they may in their inflamed or debilitated state allow tubercle bacilli to pass through into the lymph vessels beyond. Many authors have called attention to tuberculosis of the tonsils, Cohnheim and Weigert in 1884 being the first. Schlenker in

1893 established the correlation between tuberculosis of the tonsils and tuberculous cervical lymph nodes. In 1894 Lermoyez showed that there are two types of adenoid tuberculosis, one macroscopic and the other recognized only by the microscope. Dieulafoy in 1895 emphasized the frequency of latent or larval tuberculosis of all three tonsils, as determined by animal inoculation. Straus, Wright and others have found tubercle bacilli in the nasal fossæ of healthy individuals. There is no doubt that the bacilli can pass through the mucous membrane without producing any lesion. Cornet, cited by Dowd, brushed tubercle bacilli on the nasal mucous membrane of healthy animals and later found enlarged tubercular cervical lymph nodes, the mucous membrane showing no lesions. Adenoids and large tonsils, by causing mouth breathing and stagnation of the nasal mucus, predispose greatly to tuberculosis of the tonsils and to tuberculosis and suppurative inflammations of the cervical lymph nodes. Though the faucial tonsils may occasionally become infected by tubercle bacilli in the milk or in the sputum (Schlenker), undoubtedly the infection is almost always from the air, either inspired through the nose or inhaled through the mouth.

LATENT TUBERCULOSIS OF THE LYMPH NODES IN INFANTS.

Recently the subject of latent, or as the French call it, larval tuberculosis has engaged a great deal of attention, especially since von Behring's pronunciamento that tuberculosis in adults is a development of the latent tuberculosis acquired from the alimentary tract in infancy. Allan MacFadjen and Alfred MacConkey (*British Medical Journal,* July 18, 1904, page 129), in the experimental examination of mesenteric lymph nodes, tonsils and adenoids, pulverized and injected the mesenteric lymph nodes from twenty-eight children. Eight of these children had various forms of tuberculosis, but the other twenty had none; none of the twenty-eight had any intestinal symptoms. Of the twenty autopsied with no tubercular foci found, there were results by the gland inoculations from five children (6 mos., 6 mos., one stillborn, one 2½ years, and one 8 years old). In two of these tuberculosis was demonstrated by microscope; so that only in three of these was the tuberculosis truly latent. Their investigation of tonsils and adenoids yielded no positive results. Of greater interest, however, in connection with cervical tuberculosis is the fact that extension to the lungs or other organs occurs in over one-fourth

of those cases in which the lymph nodes are not removed. Why may not many cases of lung tuberculosis arise from latent—not evident—tuberculosis of the cervical lymph nodes?

Harbitz, of Kristiania, in a very interesting and careful monograph, recently published, reviews the literature to 1905 and submits the results of his own investigations of lymph node tuberculosis in children. He makes a particular study of latent tuberculosis, meaning by this the presence of tubercle bacilli in the lymph nodes without any macroscopic or microscopic evidence that they are thus infected. The discovery and proof of the larval bacilli are made by inoculation into guinea pigs. Out of 142 necropsies on children 73 were proved by absence of anatomical changes and by negative inoculation experiments to be free from any infection by tubercle bacilli. Sixty-nine had tuberculous infection, but of these 18 had the infection in latent form in the lymph nodes, discoverable only by inoculation. Of these 18 cases of latent lymph node tuberculosis, 10 occurred in infants under one year; the youngest was one month, twenty-two days, and 5 others were six months old or younger.

As to the particular groups of lymph nodes involved, in 13 out of the 18 cases the cervical chain was infected, and as has been noted heretofore, 10 of these cases were one year old or younger. The occurrence of so many instances of latent lymph node tuberculosis, demonstrable only by inoculation, in this Norwegian series calls attention to the need of more careful study of our cases in this country.

Notwithstanding these frequent findings of tuberculosis in infants, Harbitz is careful to add that enlargement of the cervical and other lymphatic nodes, due to chronic pyogenic infection, is more common in children than is their infection by the tubercle bacillus.

DISCUSSION.

DR. KOPLIK.—I am inclined to believe that the tonsil is not such an infrequent source of infection. Some years ago I published the case of a child of nine months who had been in contact with a grandfather, a tuberculous subject, and developed a tuberculosis of the tonsils and lymph nodes. I was surprised a year or so ago by the autopsy findings in a case of von Jaksch anemia, in which the terminal infection was tuberculosis; there was tuberculous adenitis and the tonsils were the seat of tuberculous infection.

DR. COTTON.—Within the past three months I was called to see a baby six months old, at the breast, that had a little temperature and restlessness. In examining the throat I noticed considerable hyperemia and a little swelling of the faucial tonsils. I had a smear made and left some laxative medicine. The case fell into the hands of another physician. Ten days later when again called I found that the child had had some temperature ever since I had seen it. The lymph nodes below the angle of the jaw, which had shown very little involvement at first (the smear showed nothing but staphylococci), had become about as large as an English walnut. It seemed rather hard, somewhat tender and the child was fretful. Within a week, three weeks from the first visit, I noticed a little softening. At that time I was going out of the city and called my assistant to watch the case. He made a bacteriological examination and demonstrated it to be tuberculosis without question. It was obstinate in healing and the discharge became somewhat chronic. There seemed to be a gland deeper which had not broken down. The edges of the wound everted, kept open and formed a keloid around it. The fistula finally closed completely some six weeks later. The child meanwhile maintained its weight very well and cut two teeth.

DR. J. H. HESS (guest), Chicago, passed around two photographs showing primary infection of the bronchial lymph nodes, with secondary involvement of three or four lumbar vertebræ.